DEC 23 1929

VOLUME 38 NUMBER 6

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

EDITORIAL BOARD

CLIFFORD G. GRULEE, Chicago

OSCAR M. SCHLOSS, New York
FRITZ B. TALBOT, Boston
H. F. HELMHOLZ, Rochester, Minn.
W. McKIM MARRIOTT, St. Louis
L. R. DeBUYS, New Orleans
JOHN C. GITTINGS, Philadelphia

DECEMBER, 1929

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $5.00

Entered as Second-Class Matter, January 4, 1911, at the Postoffice at Chicago, Illinois, Under Act of Congress of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, authorized on June 14, 1918.

Copyright, 1929, by the American Medical Association

American Journal of Diseases of Children

VOLUME 38 DECEMBER, 1929 NUMBER 6

THE DIAGNOSIS OF TUBERCULOSIS OF THE TRACHEOBRONCHIAL GLANDS *

P. ARMAND-DELILLE, M.D.

AND

C. LESTOCQUOY, M.D.

PARIS, FRANCE

The diagnosis of tuberculosis of the tracheobronchial glands is of the greatest importance, because the entire future of the child depends on it. It may make necessary open air or even preventorium life and a careful attention to diet, because if one is not careful, the child may develop generalized tuberculosis or tuberculous meningitis. Therefore, a great responsibility rests on the physician to determine whether the child has or has not tuberculosis of the tracheobronchial glands.

The importance of familial contagion of tuberculosis was emphasized, in 1902, by Grancher, who formed for that purpose the "Oeuvre de preservation de l'enfance contre la tuberculose." At that time, many physicians were not convinced of it, but now every pediatrician, in fact every physician, is convinced of the dangers of the germ-carrier parent to the children. Because of this, almost all pediatricians in the dispensaries constantly look for the disease in children, especially if one of the parents is tuberculous. As a matter of fact, this idea must serve as a basis of control for the diagnosis of tuberculosis in tracheobronchial glands.

We shall consider here only the large caseation of these glands as described by Barety, leaving out of consideration the small lesions, which may be discovered in adults, and which may follow the ordinary slight infection met with in any person living in a city. Leon Bernard rightly contrasted them with the massive, repeated, prolonged contamination by familial contact.

One should not make the diagnosis of tuberculosis of the tracheobronchial glands, as a rule, if only a positive Pirquet reaction (cutireaction) and more or less confirmed clinical and roentgenologic signs are present, and all the members of the family and those surrounding the family, including servants and nurses, are healthy. On the contrary, one must strongly suspect it if the child has been in close contact with tuberculous parents or other people, and one must search systematically at the same time for the primary focus.

Pediatricians have been too inclined to make the diagnosis of this disease on the sole basis of disputable roentgenologic interpretations.

* Submitted for publication, July 5, 1929.

The extension of the organization for child welfare, with an enlarged number of preventoriums and summer camps, has produced a system of selection based on symptoms which have not been anatomically controlled. It has been done in France, and we think also in the United States. Bronfin recently reported a study of the preventorium child among 300 children. He proved that many children have been sent to preventoriums with a diagnosis of tuberculosis of the tracheobronchial glands, without presenting any real symptoms; moreover, in those cases in which a tracheobronchial gland existed without a doubt, a former diagnosis had not been previously made. Stoloff, reporting on 6,300 patients examined in the Mount Sinai Hospital, considered that for the diagnosis of this disease there are only two means of any value, e.g., roentgenologic investigations and Pirquet reactions.

VALUE OF SIGNS POINTING TO TUBERCULOSIS OF THE TRACHEOBRONCHIAL GLANDS

Under these conditions, one is forced to determine the value of the various signs which point to the diagnosis of tuberculosis of the tracheobronchial glands by the observations at autopsy (transverse sections of the lung and of the mediastinum).

We have studied only those cases in which it seemed likely that tuberculosis did exist because there had been familial contact, and in which we have been able to recognize by clinical evidence that our presumptions were justified. In these cases there existed marked caseation of the glands, like that which Barety first described (we shall not here detail his description). We present only one type, with photographs of one plane section obtained by the method of frontal sections of the thoracic contents hardened into a block in normal position (which we have published in another [1]).

Many authors have stressed, perhaps too much, the general symptoms that lead to the diagnosis of pretuberculosis. Professor Leon Bernard justly said that pretuberculosis does not exist; a child is or is not tuberculous. The symptoms are: delicate body build, muscular weakness, narrowness of the chest, winged scapular, delicate features with long lashes, blue conjunctivae and so on. All these symptoms may be present, but one does find children apparently in perfect health who present tuberculous tracheobronchial glands. On the other hand, there are many children who have straight chests, delayed growth of the bones and muscles and poor nutrition who are not tuberculous.

A long period of slight fever must also be considered suggestive if there has been familial contact, but it may also be due to subacute

1. Armand-Delille, P.; Lestocquoy, C., and Vibert, J.: Une méthode de contrôle anatomique des radiographie pulmonaires, Presse méd. **37**:504 (April) 1929.

rhinopharyngitis. If there is no tuberculosis in the environment, these symptoms lose most of their value. Gueneau de Mussy and Barety described functional and physical signs with much detail. (The cough simulating whooping cough may be observed in adenoiditis.) In criticism of their descriptions, however, one must conclude that for the question of functional symptoms only two have any value: (1) roar-

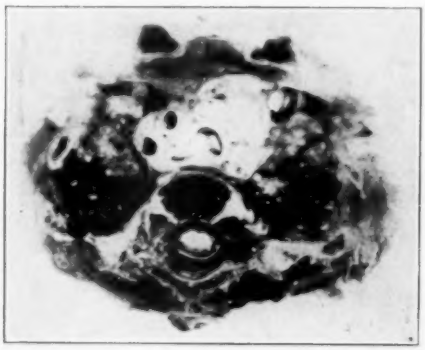


Fig. 1.—Horizontal section of the thorax through the level of the third dorsal vertebra and the sternoclavicular articulations.

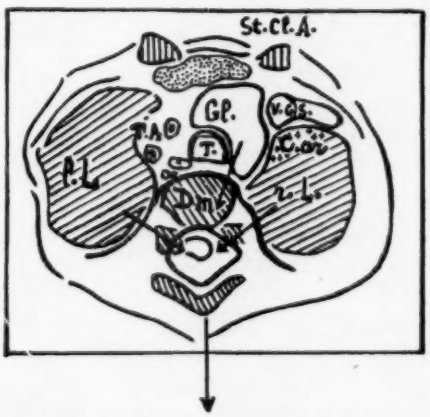


Fig. 2.—Horizontal section of the thorax through the level of the third dorsal vertebra and the sternoclavicular articulations. *Gl.* indicates the right laterotracheal ganglion enlarged and caseous. *C.ar.*, a caseous area, *r.L.*, in the anterior edge of the right lung, conceals the ganglion which is separated from the sternocostal wall by the superior vena cava (*v.c.s.*). *T* indicates the trachea; *Oe*, the esophagus, and *T.A.*, the arterial trunks. The arrows indicate the component points of percussion on the vertebral column.

ing and (2) bitonal cough (described by Marfan). As a rule, these symptoms are infrequent and may be observed only in infants or children under the age of 2 years.

One should not be surprised that the swellings of the glands do not invariably give any important signs. In certain cases, a large aneurysm of the aorta remains latent and is discovered only by roentgenography. As for physical signs, under anatomic control made by horizontal sections of the thoracic contents fixed in a block, we have been led to conclude as follows: Percussion is not able to show symptoms of enlargement of the glands, and the dulness behind the manubrium sterni, as described by the classics, cannot be really produced by the enlarged glands. There is a zone of pulmonary tissue between the glands and the wall, and if one finds parasternal dulness, it is produced by lesions of the subadjacent pulmonary parenchyma. For the posterior zone, the sound of percussion on the spinous processes is not

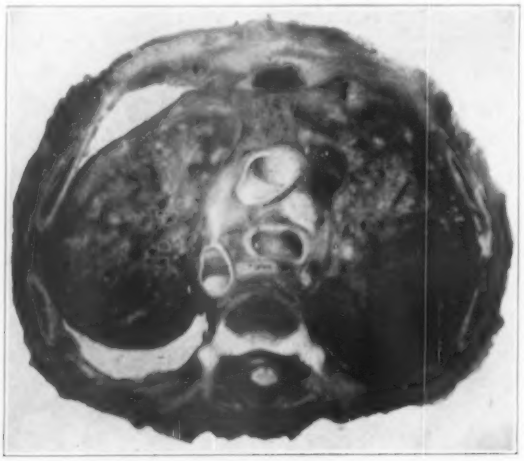


Fig. 3.—Horizontal section of the thorax through the level of the fourth dorsal vertebra.

produced by the organs of the mediastinum, but mostly by the pulmonary parenchyma, which occupies the costovertebral angle. When examined in sections, this part of the lung has a greater importance than one imagines.

We have never been able to recognize an interscapular vertebral dulness which was produced by caseous tracheobronchial glands, and we must add that in the cases in which we found caseous glands it has never been possible to discover any dulness. Auscultation also does not give symptoms of real value. The interscapulovertebral souffle, described by Gueneau de Mussy, may be observed in normal children, as many French authors have insisted (Comby, Guinon, Ribadeau-Dumas, in 1912,[2] and more recently R. Debré and Mademoiselle Petot).

2. Ribadeau-Dumas: Bull. Soc. de pédiat. de Paris **14**:442, 1912.

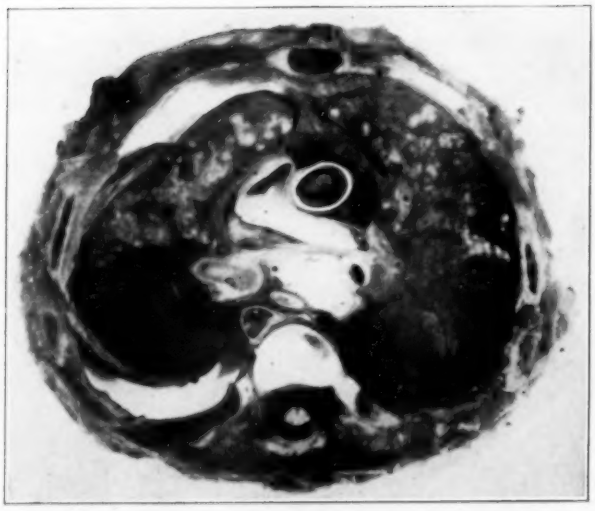


Fig. 4.—Horizontal section of the thorax passing through the intervertebral disk between the fourth and fifth dorsal vertebrae.

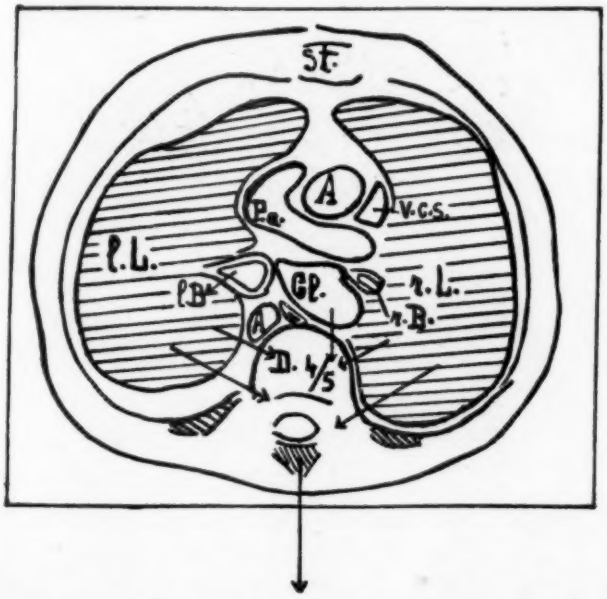


Fig. 5.—Horizontal section of the thorax passing through the intervertebral disks between the fourth and fifth dorsal vertebrae. *Gl.* indicates the caseous intertracheal bronchial ganglion; *l.B.*, the left bronchus; *r.B.*, the right bronchus; *P.a.*, the pulmonary artery, and *A*, the aorta. The vertebral sound of percussion is procured with much more resonance over the lungs than over the ganglion *Gl.*

The intensity of the souffle, more or less, depends for the most part on the conformation of the chest and the individual tone of the voice. However, one is allowed to consider that if this souffle extends below the spinal process of the fifth or sixth dorsal vertebra (which is seldom), it may correspond to a large caseous interbronchial gland and may also mean pulmonary consolidation in the vicinity.

The D'Espine sign, described by its author, has appealed greatly to pediatricians, but it seems that many physicians exaggerated the value that the author attributed to it. It seems to us that in many thin children one may find the whispered tone of voice at the spinous

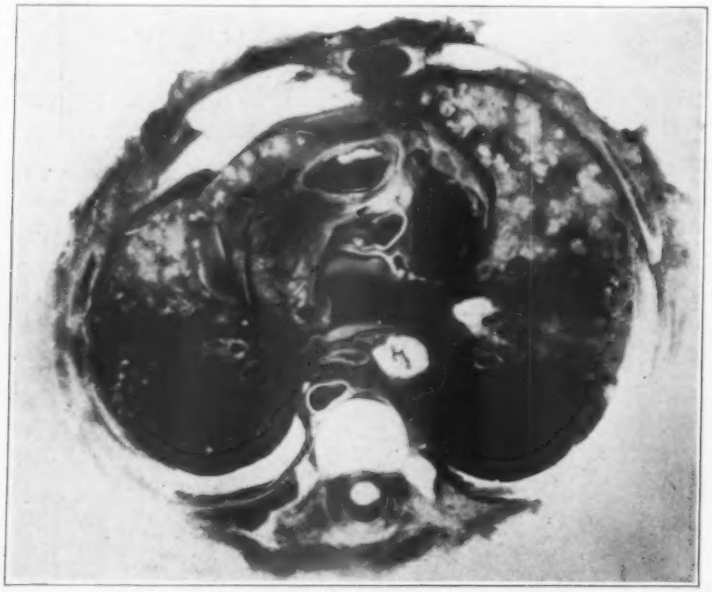


Fig. 6.—Horizontal section of the thorax through the level of the intervertebral disk between the fifth and sixth dorsal vertebrae.

process of the fourth vertebra. As to caseous glands, one knows that this observation has value only if found below the level of the fifth or sixth dorsal spine.

ROENTGENOGRAPHY AND ITS INTERPRETATION IN DIAGNOSIS

We have come to the conclusion that only instantaneous roentgenography should be used, and pictures should be taken in the frontal and lateral positions. Only thus can one make a true diagnosis. The first conditions are that these x-ray films be taken with exact technic, and then interpreted by physicians who have made pathologic controls in similar cases.

Fluoroscopy, although it allows one to recognize important changes, is not sufficient. Only a roentgenogram well taken enables one to decide, because it gives precise contours and may be compared with the section taken at autopsy.

If one would have a correct interpretation of these roentgenograms, one must first know exactly what kind of shadow roentgenography of a normal thorax gives. This has been described by Duhem and Chapron and by others.

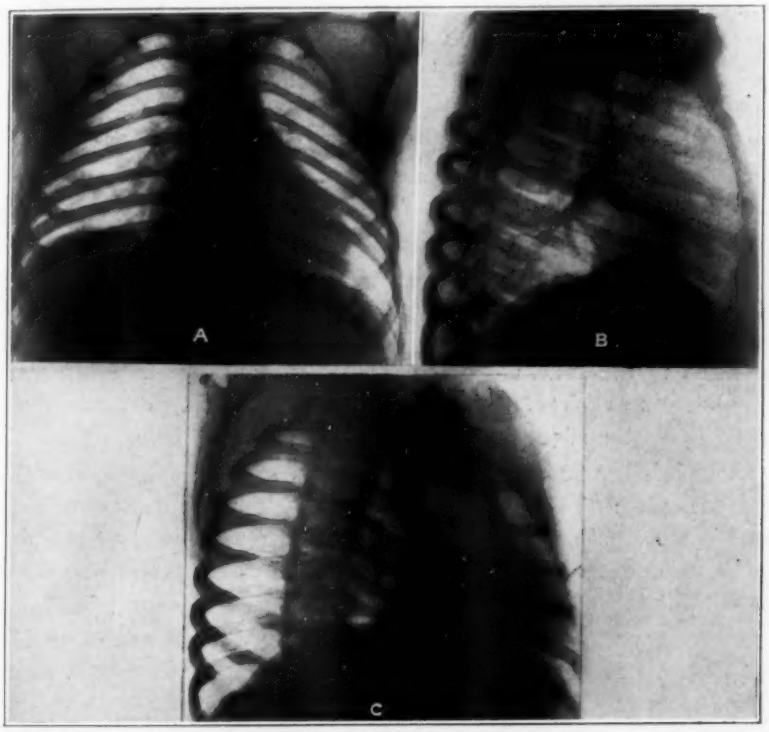


Fig. 7.—*A*, thorax of the normal child, anteroposterior view; *B*, lateral view (note that this is absolutely a straight lateral); *C*, oblique view.

They have insisted on the value of normal juxtacardiac shadows. These shadows have the shape of a horn, of external convexity, and are separated from the shadow of the heart by a clear space produced by the bronchi. They are caused by the ramifications of the trunks of the pulmonary arteries. On the left they are entirely uncovered; on the right they are partially covered by the shadow of the heart. The ramifications of the pulmonary artery leaving the hilum and crossing

the ramifications of the bronchi give a confused shadow of small spots which must not be confused with small calcified glands. We wish to emphasize that if a film is taken in the right oblique anterior position, as is usually done, one obtains a rather large indistinct shadow at the bifurcation of the trachea. As Lestocquoy and Lehmann [3] demonstrated, these shadows are caused by the right branch of the pulmonary artery and the large right pulmonary vein in the left auricle.

This is why roentgenography in the right oblique anterior positions is not satisfactory, because this shadow may lead one to think that there is an adenopathy which does not really exist. Under these conditions, it seems better to use roentgenograms taken in profile; that is, by lateral roentgenography. This method on the normal subject, as Lestocquoy and Lehmann demonstrated, offers the advantage of showing the clear posterior mediastinal space in its largest diameter. On the other hand, the vascular shadows of the big trunk are found on the anterior border, and are blended with the shadow of the heart. The only shadows projected are those of the ramifications of the arteries and pulmonary veins which give lineal shadows easy to interpret.

The upper part of the profile roentgenogram (lateral view) of the normal subject shows plainly the clearness of the trachea, which descends to the fourth dorsal vertebra, where there is a bifurcation. One also sees clearly the right bronchus, which descends nearly vertically with a slight obliquity toward the front.

The left bronchus, on the contrary, is slightly oblique, downward and backward. This interbronchial space does not show any notable shadow. Beneath this region at the level of the fifth or sixth dorsal vertebra one sees the paint-brush appearance on the vascular ramifications.

If caseous glands exist, the roentgenograms (frontal and lateral) show ramifications which, carefully analyzed, allow one to arrive at a precise diagnosis, as has been demonstrated by anatomic control in a number of cases. In the typical cases, in the frontal view, there is a rounded shadow with distinct edges, which overlaps by about 1 cm. the shadow of normal concavity produced by the superior vena cava and innominate trunk. In height, its upper extremity reaches the shadow of the clavicle; the lower extremity descends to the level of the junction of the cardiac edge and the shadow of the superior vena cava.

Inside the border of the shadow of the lymph node is the tracheal clearness; this tracheal clearness is usually central but may be deviated

3. Lestocquoy, C., and Lehmann, R.: Presse méd. **37**:293 (March) 1929.

or contracted, as Marfan and Barret showed first in France. Bronfin [4] reproduced a characteristic film in figure 8 of his pamphlet.

In less typical cases, there is only an enlargement of the shadow of the pedicle of the heart on the right side, with indefinite contour, which shows that lesions exist in the surrounding parenchyma in the anteroposterior views which are confused with the shadows of the lymph nodes.

In the discussion of these aspects, one must be careful not to fall into the common error since an enlargement of this pedicular shadow may be due to the child's being slightly turned from the roentgen-ray apparatus or because the picture was not perfectly centered. This

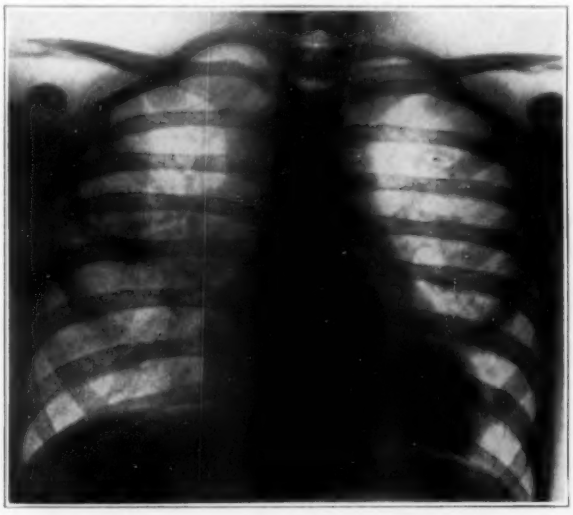


Fig. 8.—Frontal roentgenogram of the right laterotracheal gland and interbronchial gland caseation.

shows that it is necessary to take a roentgenogram at a distance of 1 meter to reduce to a minimum the distortion of the shadow of the heart.

The roentgenogram must be made instantaneously (fraction of a second) to obtain clear outlines.

The left laterotracheal group, always smaller than the right, is hidden by the left aortic shadow, which it overlaps only in exceptional cases. One sees then a round shadow immediately over the shadow of the aorta and the pulmonary artery. The intertracheobronchial group which lies in the angle of the bifurcation of the trachea, although it is the largest, cannot, as a rule, be detected by this anteroposterior view.

4. Bronfin, I. D.: The Preventorium Child: A Clinical Review of Three Hundred Cases, Am. J. Dis. Child. **36:**931 (Nov.) 1928.

The right and left bronchial groups give different shadows. The right interbronchial group is clearly, though incompletely, visible, as the most internal parts are more or less masked by the cardiac shadow. As a rule, however, one may find its internal limit if one is able to recognize the clearness of the right bronchus, which is nearly always visible in or through the cardiac shadow.

In certain cases, when the glands are voluminous, they give a shadow the size of an almond. In less clear cases, the shadows of the lymph nodes are superimposed on the vascular shadow; when they are not

Fig. 9.—Lateral roentgenogram of caseated intertracheobronchial glands. Compare with anatomic control in figure 10.

calcified or enormous, it is difficult to avoid the erroneous interpretations which have been and still are frequent.

The left bronchial group is almost completely invisible, a fact which can easily be understood if one examines a roentgenogram after the injection of iodized oil. One sees that the left bronchus divides into ramifications behind the cardiac shadows, and it will be seen later that, as has been described, the perihilar shadows are in reality the shadow of parenchymatous lesions of the posterior part of the lung.

The purpose of the lateral roentgenogram is to examine the intertracheobronchial group, which is not visible on anteroposterior roentgenograms. In the typical cases, it gives a rounded or oval shadow with

regular edges, the size of an almond, directly under the tracheal bifurcation. This shadow occupies the posterior area of the mediastinal space, which is really the posterior roentgenologic gland area. On the oblique view, one can also find this shadow, but we have seen that in the normal subject the posterior mediastinum is occupied by a vascular shadow.

In the upper part of the mediastinal space, the tracheobronchial glands, which lie on both sides of the trachea, project rounded shadows which partly overlap the tracheal clearness. They should not be confused with the superior vena cava which lies more in front.

Fig. 10.—Oblique section of the thorax passing through caseated intertracheobronchial glands. Compare with roentgenographic control of figure 9.

We must also note that the shadows given by the left bronchi are important, especially in connection with the glands of the left hilum.

It is evident that it is useless to examine a lateral roentgenogram without the anteroposterior roentgenogram. One must first examine carefully the normal roentgenograms, which show that lesions of the parenchyma give no shadows in the area adjacent to the mediastinum. It should be noted, therefore, that a certain number of shadows which have been described as perihilar are in reality, as anatomic control has shown, made by parenchymatous lesions.

CONCLUSION

The caseated tracheobronchial glands in infancy and childhood are difficult to diagnose. The physical signs, such as dulness and D'Espine signs, are not constant; the Pirquet test is also not sufficient. The only way to arrive at a proper diagnosis is to supervise all children who have been in frequent and close contact with a germ carrier, and to have good roentgenograms (frontal and lateral) carefully examined, comparing them with films from other cases in which roentgenographic observations have been checked at autopsy.

TUBERCULIN SKIN REACTIONS*

CHARLES HENDEE SMITH, M.D.
NEW YORK

In 1890, Koch introduced the general or subcutaneous tuberculin reaction. In 1902, Branner noted a local skin reaction around the site of therapeutic injections of tuberculin. Pirquet [1] described his cutaneous scratch test in 1907; in the same year Wolff-Eisner and Calmette brought out the conjunctival test, and Morro introduced the percutaneous or inunction test. In 1908, Mantoux [2] described the intracutaneous test. In 1909, Hamburger and Monti published the results in a series of cases tested with the Pirquet, followed by the "Stich" reaction. No noteworthy additions have been made to this list in twenty years. In all these tests the essential principle is the same, the object being to bring tuberculin into contact with the deeper epithelial cells. The success of the tests depends entirely on the accuracy with which this contact is established, and therefore the Mantoux and Stich reactions should be expected to be more certain than the Pirquet and Morro.

A positive tuberculin reaction means that the tubercle bacillus has lived and grown in the body. The best explanation for the reaction is that cells which have once been dosed with tuberculin from living tubercle bacilli develop the power to react when tuberculin is again brought into contact with them. It is said to be possible to cause a general anaphylaxis by reinjecting tuberculin into animals, but the skin sensitivity which gives the reaction has never been produced this way. The skin reaction allergen is probably intracellular, bound with the tissues. It is possible to sensitize local areas of skin by repeated tuberculin injections, but the rest of the skin of the body will not react [3] (fig. 1). General skin sensitivity is seen only after the tubercle bacillus has lived in the body, that is, after true tubercle formation.

---

* Submitted for publication, June 18, 1929.

* From the Department of Diseases of Children, Columbia University, College of Physicians and Surgeons and Children's Medical Division, Bellevue Hospital.

* Read before the American Pediatric Society, June 2, 1926.

1. Pirquet, C.: Demonstration zur Tuberculin Reaction, Berl. klin. Wchnschr. **44**:699, 1907.

2. Mantoux, C.: Intradermo reaction de la tuberculin, Compt. rend. Acad. d. sc. **147**:355, 1908.

3. Kolmer, J. A.: Nature of Tuberculin Reaction, Therap. Gas. **46**:381, 1924.

TECHNIC

On account of the ease with which it is made, the Pirquet (epidermal) test has been most used in all countries as a routine procedure. The Mantoux, or intracutaneous, test was not generally accepted as an improvement on the Pirquet, and at first was little used in this country, although many writers have called attention to its

Fig. 1.—A marantic child with some symptoms suggesting tuberculosis. The Mantoux reaction in the left arm was negative with a dosage of 0.05 and 0.2 mg., and positive with 0.5 mg. in the same arm. The large erythema was followed by desquamation and pigmentation. These three tests were made on the same arm by mistake of a new intern. The reaction was negative with 0.5 and 1 mg. in the right arm. Autopsy revealed no evidence of tuberculosis.

greater accuracy. During the past few years, it has come into more general use.

The main advantage of the Pirquet test is the simplicity of the technic. The skin is cleansed with alcohol, and a small area of epidermis

is removed, either a linear scratch being made with a needle or a circular one with a sterile toothpick or a scarifier (a small, flat, chisel-shaped piece of metal which is twisted between the fingers). It is desirable to draw no blood. Into the denuded area a drop of pure old tuberculin is rubbed gently and allowed to dry. A control scarification is made in the same way, but no tuberculin is applied. Two test scratches and one control should be made. The site is usually the anterior surface of the forearm. A dressing of gauze held with adhesive tape may be applied; this not only avoids any possible infection by scratching, but also prevents wiping off of the tuberculin.

A positive reaction begins in from twenty-four to forty-eight hours, occasionally later. The transient erythemas of the first few hours should be disregarded, since they are not the true tuberculin reactions. The reaction should be read daily for five or six days, if possible, or if this cannot be done, at forty-eight hours and on the fifth or sixth day. A positive reaction is shown by an area of erythema and edema around the original scratch. This varies from a slight redness to an area, about 1 cm. in diameter, which is bright red and raised above the surrounding skin. As the exudation into the skin subsides, there is often a deposit of pigment so that the area is stained for two or three weeks and there is frequently slight desquamation. Severe reactions occur at times, resulting in small vesicles or even areas of necrosis. I have seen a few reactions in which a large area was scratched and in which there was a real slough, a scar, and even a constitutional reaction.

The Pirquet test is easy to perform, but has distinct disadvantages. In the first place, it is not always easy to distinguish between a true reaction and that due to trauma. If the scratch is too deep, there is often a little swelling around it, which may lead an untrained observer to call the reaction positive. But a greater disadvantage is the uncertainty of the test. Just as a vaccination may not take when performed by the epidermal method, so the Pirquet test may fail because the tuberculin was not kept in contact with the cells of the deeper layer of the skin long enough to excite the specific response. The inaccuracy of the Pirquet test is shown by the fact that many children will fail to react on the first test and yet will show a positive reaction on a retest a few days later. It has been thought that this is due to the raising of the sensitivity by the first test. If this is true, the Mantoux should also give a much higher percentage of positive results on the second test, but in my experience this happens rarely. It seems more likely, therefore, that the unreliability of the Pirquet test explains the difference in the results on the first and second tests. Moreover, if two tests are made at the same time, one may fail to react although the other reacts sharply (fig. 2). If a single test is made and does not cause a reaction,

it is necessary to repeat the test once or twice to be sure that the failure was not due to technical error.

This unreliability of the Pirquet test became manifest to those in the Children's Medical Division at Bellevue Hospital about ten years ago. The Mantoux reaction was taken up in the hope of finding a more accurate test. Immediately it was found that many patients reacted definitely to the Mantoux test and failed to react to the Pirquet, although two of the latter tests were always done. This difference

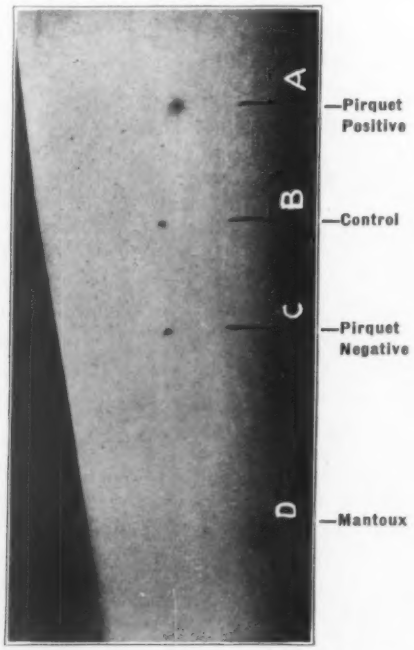


Fig. 2.—Pirquet reactions and control. *A* indicates a positive Pirquet; *B*, a control; *C*, a negative Pirquet and *D*, a Mantoux reaction. The latter gives a larger erythema and is much more easily read. This shows the unreliability of the Pirquet test.

persisted year after year and at about the same rate, i. e., the Mantoux test giving about twice as many positive reactions as the Pirquet. For several years both tests were performed on all children, but the Pirquet was finally dropped as a waste of time because of its uncertainty.

The Mantoux or intracutaneous test is performed by injecting into the epidermis a dilution of old tuberculin in salt solution. A small wheal must be made in the skin itself. This reaction appears in from twenty-four to forty-eight hours. The test should be read always after forty-eight hours and daily for five days if possible, or at least on the

fifth to the seventh day. The reaction is of the same nature as the Pirquet but is much more definite, the wheal being usually 1 cm. in diameter, frequently larger. Severe reactions may have a central raised area from 3 to 5 cm. across and a surrounding areola from 8 to 12 cm. in diameter. Occasionally there is vesiculation and rarely a slough (fig. 3). At times a red streak is seen following the lymphatics up the the arm (fig. 4). The Mantoux test is much more certain than the Pirquet, because the tuberculin is injected into the exact layer of cells which react and cannot be wiped off as in the epidermal method.

The dilution used must be made up accurately. After trying various dilutions, my co-workers and I have found that 1:1,000 is the most satisfactory. Weaker solutions have no advantage and stronger ones

Fig. 3.—A sloughing ulcer resulting from a Mantoux test in a negro with bone and lymphatic tuberculosis. The dose administered was 0.1 cc. of a 1:1,000 solution of tuberculin, or 0.1 mg. This type of reaction has occurred only three or four times in more than 10,000 cases.

are not without danger, because it is possible to give enough tuberculin with them to cause a general febrile reaction. This happened to one child when several different strengths were tested to determine whether there was a quantitative factor in the reactions. The child received 0.1 cc. of each of three solutions (1:1,000, 1:2,000 and 1:3,000), which gave approximately 0.18 mg., and this was enough to excite a general reaction. Since then more than 0.1 mg. has never been given as an initial dose, and no general reactions have resulted.

In retesting cases in which the first test is negative a stronger solution may be used in order to give the desired dose with a small wheal (1:200, 1:100 or 1:10). These strong solutions should never be used for the first test.

The 1:1,000 solution makes the calculation of the dose easy, and this is important because medical men are not, as a rule, reliable arithmeticians: 0.1 cc. of 1:1,000 equals 0.1 mg.; 0.05 cc. of 1:1,000 equals 0.05 mg. and 0.5 cc. of 1:1,000 equals 0.5 mg., etc.

The dilution may be made up with no great difficulty. The old tuberculin must be accurately measured with a pipet or with a tuberculin syringe. One-tenth cc. of old tuberculin in 100 cc. of salt solution gives 1:1,000 solution. This can be made easily in the hospital where sterile salt solution is readily available. But it is a simple matter, even in the physician's office, to boil 100 cc. of water, add a salt tablet

Fig. 4.—A vesicular Mantoux reaction with a large area of erythema and a red streak up the lymphatic vessel. The lower area is pigmentation from a test made ten days before.

and 0.1 cc. of tuberculin. The solution is ready when cool and the unused portion can be thrown away. To obviate even this amount of trouble and to make the test readily available at the bedside, in the office or hospital, the Health Department of New York City, at my suggestion, has put out an outfit similar to that used for the Schick test.[4] It consists of a capillary tube containing approximately 0.01

4. The Lederle Laboratories also make a similar outfit but it has two bottles—one containing a small amount of tuberculin diluted with glycerin, the other about 1 cc. of salt solution. This has a possible disadvantage in the dilution of the old tuberculin. The "Mantoux" outfit of the Lederle Laboratory is a 1:10,000 dilution, but they also have a 1:1,000 solution, or the therapeutic dilution "C" may be used.

cc. of old tuberculin and a vial of 10 cc. of salt solution. This removes the one objection to the Mantoux test, i. e., the trouble of making up the dilution.

The diluted solution should be kept cold and should not be used after it is one or two days old. It does not always deteriorate if kept on ice, but may do so at room temperatures.

The dilution of 1:1,000 has been found to be the most practical one for general use by many others (Bass,[5] Elliott,[6] Krogsgaard[7] and others). The dose 0.1 cc. (0.1 mg.) is the amount used for the initial test as a rule. This amount does not cause a general reaction but may produce a severe local one in a sensitive case. For that reason a smaller initial injection, 0.05 cc. (fig. 5) is safer, and the routine in use

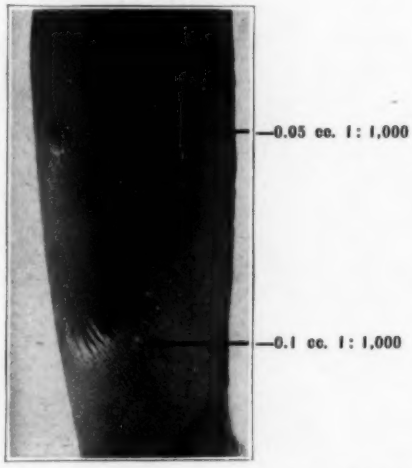


Fig. 5.—Mantoux reactions on the arm of a negro who had extensive cervical adenitis. The upper area shows the reaction following 0.05 cc. of a 1: 1,000 solution of tuberculin, or 0.05 mg.; the lower area that following 0.1 cc. of 1: 1,000 or 0.1 mg. The reactions were severe and vesicular. This shows the importance of small doses in cases of probable massive tuberculosis.

at present at Bellevue is as follows: first test 0.05 cc. of 1:1,000 solution or 0.05 mg. in left arm below elbow; second test 0.2 of 1:1,000 solution or 0.2 mg. in mid-forearm; third test 0.1 cc. of 1:200 solution or 0.5 mg. above wrist; fourth test 0.2 cc of 1:200 solution or 1.0 mg. in

5. Bass, M. H.: Cutaneous and Intracutaneous Tuberculin Tests in Infancy and Childhood, Am. J. Dis. Child. **15**:313 (May) 1918.

6. Elliott, J. H.: Survey of Ontario Children, Tubercle **7**:122, 1925.

7. Krogsgaard, H. R.: Étude sur la reaction Mantoux, Acta pediat. **5**:103, 1926.

right arm below elbow and fifth test 0.2 cc. of 1:100 solution or 2.0 mg. in mid-forearm.

As a rule, only two tests are made, except in cases in which there is some reason to suspect that there is tuberculosis present with a lowered sensitivity of the skin, e. g., during measles and many other febrile diseases, with an overwhelming infection, etc. As a matter of fact, it is a rare occurrence to obtain a positive reaction on the second or later tests when the first is negative. Occasionally, the first test has been called negative when the reaction was not marked, when made on the skin of a negro or when the staff man who recorded the reaction was inexperienced. In many instances when the second test is positive, it is possible to go back to the original test and find pig-

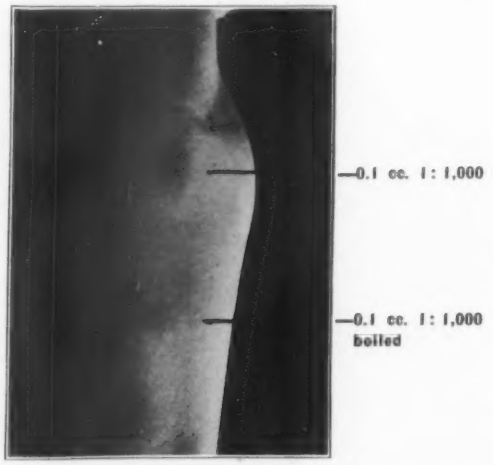


Fig. 6.—Mantoux reactions. The upper area shows the reaction following the use of 0.1 cc. of a 1: 1,000 solution of tuberculin, or 0.1 mg.; the lower area, that following the same dose of solution boiled twenty minutes. Note that there is practically no difference in the reaction.

mentation, showing that it was really positive. The later tests have been positive only a few times each year, and then in cases in which by tuberculosis has been pronounced or occasionally following an acute febrile illness during which the first test was negative. For these reasons, it seems fair to assume that the Mantoux is reliable, and that a negative first test usually means that tuberculosis is not present.

A control to the Mantoux test is not necessary. Salt solution or other inert solutions never cause a reaction which may be confused with that due to tuberculin. In order to be certain that the broth element in the old tuberculin could not cause a reaction, a solution made

up by the New York City Department of Health through the kindness of Dr. Krumwiede was used as a control in all cases, over a period of one year. This was made from the same culture medium used in the manufacture of old tuberculin and was treated in exactly the same way, except that no tubercle bacilli were grown on it. Dilutions of this concentrated broth were injected near the sites of the Mantoux tests. No reactions were obtained in any case and it was concluded that the control was not necessary. After about a year, three children showed reactions to the control which were almost identical with those caused by the dilute tuberculin. Careful inquiry elicited the fact that the intern had broken one needle on the morning on which these tests were made and had used the same needle on both syringes, so that some of the tuberculin was introduced in the control.[8]

Tuberculin is not thermolabile, but remains active after boiling for several hours (fig. 6). Therefore it is evident that a needle or syringe which has been used for tuberculin should never be used for a control. For the same reason, such a needle or syringe should never be used for a Schick test, because it may give a tuberculin reaction which might easily be confused with a positive Schick reaction.

There are certain sources of error in all tuberculin reactions. It was found that acute illness may suppress the reaction, especially if there is high fever, as in measles, pneumonia and typhoid, but it does not always do so. Severe tuberculosis frequently suppresses it; so a child with general miliary tuberculosis or one who is wasted to the last degree with long chronic disease may not react to the average dose. The sensitiveness is merely reduced, not absent, in such cases, for Happ and Casparis [9] were able to obtain reactions in every case of known tuberculosis by increasing the dose up to 1 mg. or more. I have had the same experience. Possible errors in technic must always be borne in mind. The tuberculin must be active, not too old, and not contaminated with

8. An experience at an out of town clinic was instructive. The tuberculin outfit was sent to a local physician several days ahead so that he could make tests to demonstrate the reaction at the clinic. On his own initiative he made controls with salt solution on all the children tested. On the day of the clinic nearly half of the control tests showed a sharp reaction, as large as the tests made with tuberculin dilution. This was puzzling and somewhat embarrassing to one trying to demonstrate the specificity of the test. However, it was found that the man who made the tests had used two syringes and a dozen needles. After each injection the needles were put back into the sterilizer and boiled, but no attempt was made to keep the needles used for the control apart from the others, so that some of the control tests were made with needles which had been used for tuberculin.

9. Happ, W. M., and Casparis, H. R.: Value of Intracutaneous Tuberculin Test in Extensive Tuberculosis, Am. J. Dis. Child. **23**:527 (June) 1922.

bacteria or molds. The test must be properly done, so that some tuberculin is left in contact with the cells of the deeper layer of the skin long enough to cause the reaction. Errors in reading must be discounted.

A preliminary Pirquet test is advocated by many. If this is negative, an intracutaneous test is made with a varying initial dose. In some hospitals a large amount (1 mg.) is used on the first Mantoux test following a negative Pirquet. This plan is not without danger, because of the unreliability of the Pirquet test. A false reading of the latter which leads to the injection of a large amount of tuberculin into an infected child is almost certain to cause a general febrile reaction or a severe local one in some cases. Furthermore, the preliminary Pirquet test wastes time in a hospital and may prevent a proper diagnosis if the

Fig. 7.—Mantoux reactions. The upper area shows the reaction following the use of 0.1 cc. of a 1 : 1,000 solution of tuberculin, or 0.1 mg.; the lower, that following 0.1 cc. of a 1 : 10,000 solution, or 0.01 mg. Note that the size of the reaction is about the same, although one solution contained ten times more tuberculin. The third area of erythema was the reaction to a Schick test.

child is removed or discharged in a few days. In private practice the preliminary Pirquet test only prolongs the anxiety of the parent and puts the physician in an awkward position trying to explain that he has used the less accurate test, expecting to follow it with the more reliable one. The parent naturally wants the correct answer quickly and can see no advantage in this plan.

Many attempts have been made to find a quantitative tuberculin reaction. The Mantoux method would seem to be the ideal one for quantitative tests, and it has been shown that in weak dilutions the test may be negative, coming out with stronger solutions and increasing in

the erythema produced as the solution is made stronger. The significance of this is uncertain, however, and the test is not always definitely quantitative. Thus I have found that, with the stronger dilutions at least, the size of the reaction seems to depend more on the size of the wheal made by the injection than on the actual amount of tuberculin injected. For example 0.1 cc. of 1:10,000 solution (0.01 mg.) usually gives about the same reaction as 0.1 cc. of 1:1,000 (0.1 mg.), while 0.01 cc. of 1:1,000 (0.01 mg.) gives a much smaller erythema than the same dose when given as 0.1 cc. of 1:10,000 solution (fig. 7 and 8).

THE INCIDENCE OF INFECTION

During the years following the announcement of the tuberculin tests, the results in many series of cases were published showing the

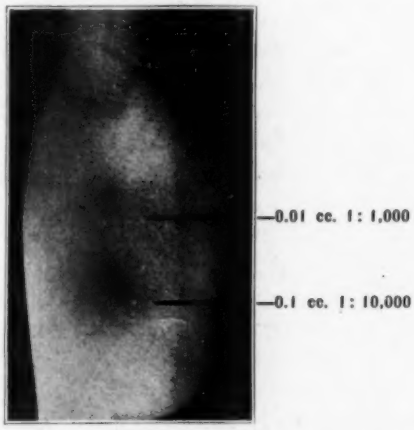


Fig. 8.—Mantoux reactions. The upper area shows the reaction following the use of a 0.01 cc. of a 1:1,000 solution, or 0.01 mg.; the lower area, that following 0.1 cc. of 1:10,000 or 0.01 mg. Note that the weaker solution gave the larger reaction because the size of the wheal made by the injection was ten times larger though the dose of tuberculin was identical.

percentages of positive reactions at different ages for the purposes of estimating the incidence of infection with the tubercle bacillus. One of the first of these was the series of Hamburger and Monti (1909) from Vienna.[10] This showed a high incidence of infection, up to 50 per cent at five years and 94 per cent at puberty, as shown by a positive Stich reaction. This test differs from the Mantoux only in the depth to which the needle is inserted, so that the results must be comparable. These figures were generally accepted as representing the condition

10. Hamburger and Monti: Die Tuberculose Haufigkeit des Kindersalters, München med. Wchnschr. **56**:449, 1909.

found all over the world. The same paper showed the relative inaccuracy of the Pirquet test, but this fact was generally overlooked, although Hamburger has repeatedly called attention to it. So the publications of the next few years reported series tested by the Pirquet method. Most of these European cities showed an amount of infection roughly parallel to that in Vienna (Jeannerett [11]).

The first series in America reported by Veeder and Johnston [12] from St. Louis, and Gittings and Donnelly [13] from Philadelphia, gave a curve which was different from those of the European cities, not over 45 per cent at 13 to 14 years. Both were based on Pirquet's tests (fig. 10). Bass [5] called attention to the greater accuracy of the Mantoux test in 1918 and reported results in a series of cases tested. In 1921 to 1925 I reported a series from the Children's Medical Division of Bellevue Hospital [14] showing about the same amount of positive infection in New York as the aforementioned series from St. Louis and Philadelphia. These figures were based on Mantoux tests, however.

The present series is based on the tests on all the children in the wards of the Children's Medical Division from Jan. 1, 1921 to Jan. 1, 1929, with the exception of those who were in the hospital too short a time to be tested and excluding one period of three months when the statistics were not kept. It should give a fair estimate of the amount of tuberculous infection present in the community because many children were admitted for minor complaints as well as for severe illness. It does include many who were admitted for tuberculosis and therefore may show a somewhat higher incidence than that of the average population. The series comprises 7,668 children from birth to 13 years of age. The percentage of positive reactions is shown in table 1 and is much lower than the early European figures.

The first six months shows about 3.38 per cent positive reactions; from 6 months to 2 years, from 8 to 12.5 per cent. The runabout age, from 2 to 6 years, gives from 15.6 to 18 per cent, but the early school years, from 6 to 10, when the contacts of the child increase, give almost double that figure, from 26.3 to 29.7 per cent. From 10 to 13 years the percentage rises to from 38.8 to 43 per cent. What it is above that age is not known, but at present adults and adolescents are being tested. On a few hundred tests we have found 90 per cent positive in adults

---

11. Jeannerett, L.: L'intraderma reaction et ses applications en médecine infantile, **33**:37, 1913.

12. Veeder, B. S., and Johnston, M. R.: Frequency of Infection with Tubercle Bacillus in Childhood, Am. J. Dis. Child. **9**:478 (June) 1915.

13. Gittings, J. C., and Donnelly, J. D.: Statistics on the Pirquet Reaction, Arch. Pediat. **38**:78, 1921.

14. Smith, Charles Hendee: Diagnosis of Tuberculosis in Childhood, Proc. Am. Ped. Soc. **33**:246, 1921; Bull. N. Y. Tuberc. A., 1923; Atlantic M. J. **28**:725, 1925.

but have not yet enough figures to report on adolescents. We have found about 16 per cent positive Mantoux reactions each year, and the curves showing the amount of infection at various ages are almost identical in each year. Since the tests are made by a changing house staff (even though under careful supervision and direction), one would expect a greater variation than has actually been obtained.

TABLE 1.—*Mantoux Reactions in Children in Children's Medical Division, Bellevue Hospital, from 1921 to 1928*

Age	Total Tested	Positive Reactions	Per Cent Positive
0 to 6 months	1,425	48	3.38
6 to 12 months	982	79	8.0
1 to 2 years	1,088	136	12.5
2 to 3 years	704	104	
3 to 4 years	402	63	15.6
4 to 5 years	421	68	
5 to 6 years	396	84	18.6
6 to 7 years	425	104	
7 to 8 years	369	105	26.3
8 to 9 years	379	95	
9 to 10 years	356	116	28.7
10 to 11 years	322	127	38.8
11 to 12 years	316	121	
12 to 13 years	83	36	43.
	7,668	1,286	

TABLE 2.—*Relative Value of Pirquet and Mantoux Tests*

Age	Total Cases	Pirquet Per Cent Positive	Mantoux Per Cent Positive
0 to 6 months	594	1.35	5.21
6 to 12 months	394	3.3	8.4
1 to 2 years	417	5.	8.6
2 to 3 years	247	4.45	11.3
3 to 4 years	202	5.95	11.9
4 to 5 years	169	5.9	14.7
5 to 6 years	146	10.25	17.7
6 to 7 years	182	13.7	26.4
7 to 8 years	132	12.8	26.2
8 to 9 years	159	8.8	25.
9 to 10 years	130	15.4	37.
10 to 11 years	153	21.	38.
11 to 12 years	132	25.8	45.5
12 to 13 years	55	(18.2)	45.5
	3112		
Total cases positive Pirquet test		242 = 7.8%	
Total cases positive Mantoux test		517 = 16.5%	

For four years of this period, two Pirquet tests were done on all children at the same time (3,112 in all). The results compared with those of the Mantoux test are shown in table 2. More than twice as many Mantoux as Pirquet tests were obtained, and the difference between the two tests was striking at all ages. This confirms Hamburger's early criticism of the Pirquet test and also that of later writers. There is a good deal of variation in the ratio of dependability of Pirquet

to Mantoux in the results of different writers. Krogsgaard [7] found only 12 per cent in favor of the Mantoux test but Hamburger [15] stated the belief that the Pirquet test is only 40 per cent reliable. Others have found a difference of from 4 to 60 per cent in the results.

Figure 9 shows curves plotted from the figures of Davidsohn [16] from Berlin, and Engel and Bauer quoted by Nothman [17] from Düsseldorf showing that in each city the percentage of positive Stich reactions was much higher than those obtained by the Pirquet test. The absolute number of positive reactions is higher than the results at Bellevue,

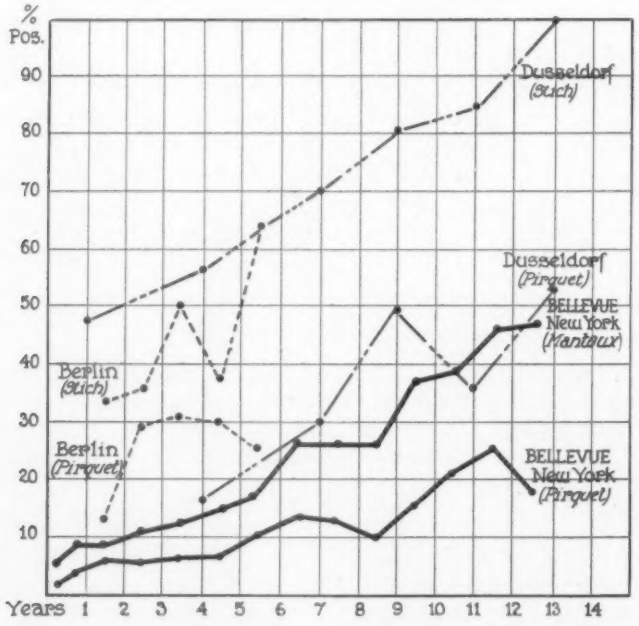


Fig. 9.—Comparison between Pirquet and Mantoux or Stich tests. The curves marked Düsseldorf are from Engel and Bauer (Berl. klin. Wchnschr. **47**:381, 1910); those marked Berlin are from Davidsohn (Ztschr. f. Kinderh. **26**:178, 1920). The curves marked Bellevue are taken from the data in table 1. Note that the positive Pirquet curves are roughly only 50 per cent of the Mantoux or Stich curves.

which are also plotted, but the relative value of the Pirquet and the Mantoux or the Stich test corresponds to what we have found.

15. Hamburger, F.: Frequency of Tuberculosis in Children, Am. J. Dis. Child. **23**:481 (June) 1922.

16. Davidsohn, H.: Ueber die gegenwartige Ausbreitung der Tuberculose und der Tuberculose Infection unter Berliner Kindern, Ztschr. f. Kinderh. **26**:178, 1920.

17. Notham: Ueber die Haufigkeit der Tuberculose im Kindesalter, Berl. klin. Wchnschr. **47**:381, 1910.

Pirquet [18] himself found nearly twice as many positive reactions on the second tests as on the first. He interpreted this as a bringing out of a latent sensitivity by the first test. It seems more likely that it merely corroborates the observations of others as to the unreliability of this test. For these reasons all series based on Pirquet tests must be disregarded as a basis for showing the incidence of infection. Most of the American statistics are open to this criticism. In addition several of the different series have a second source of error, due to the fact that only suspected cases were tested (fig. 10).

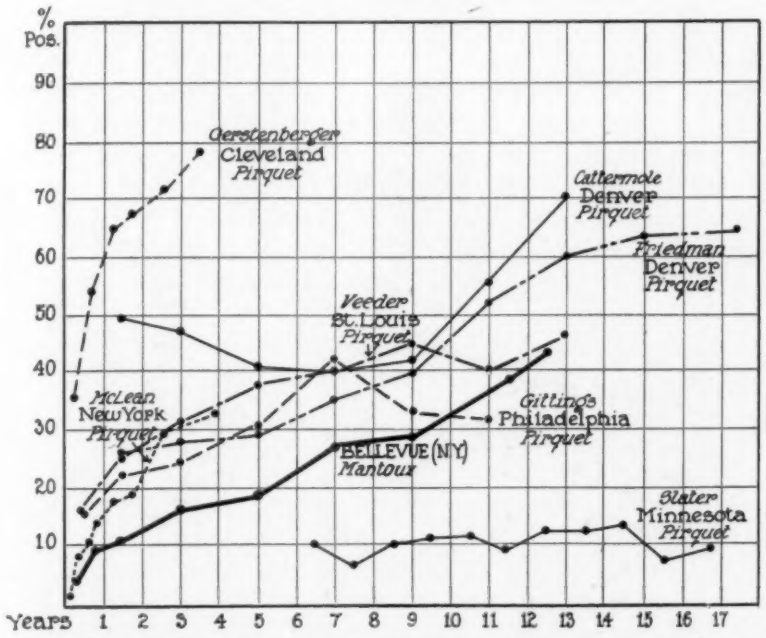


Fig. 10.—The incidence of positive Pirquet tests in various American cities. These do not represent the true incidence of infection with tuberculosis, because they were done by the Pirquet method, which gives too low results, and on those suspected of having tuberculosis or coming in contact with tuberculosis patients, so that the percentage of positive tests would be high. (See references in text for source.)

Gittings and Donnelly [13] said "As a rule the test was made on suspicious cases only."

Veeder and Johnston [12] made the statement "All children under 2 years were tested. As a rule tests are made on children over two only when there is some indication."

18. Pirquet, C.: Frequency of Tuberculosis in Children, J. A. M. A. **52**:675 (Feb. 27) 1909.

McLean and Jeidell [19] stated that "As a rule the test was made on suspicious cases only." Only 49 per cent of the patients were tested in this series from the Babies' Hospital (New York).

Wahl and Gerstenberger [20] asserted that "All negative reactions were taken into account except those occurring in patients who gave a negative history of contact and who were free from tuberculosis clinically."

Cattermole [21] and Freedman [22] from Colorado tested children, many of whom were in contact with tuberculous parents or relatives.

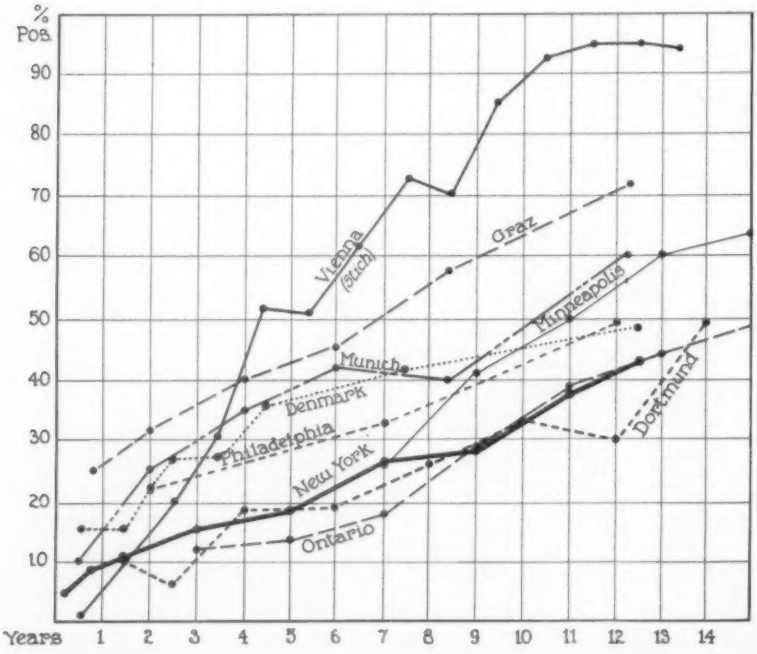


Fig. 11.—Curves marked with names of cities showing incidence of positive Mantoux tests by years. The source is as follows: Vienna, Hamburger and Monti (München med. Wchnschr. **56**:449, 1909); Graz, Barchetti (Arch. f. Kinderh. **71**:180, 1922); Munich, Barchetti; Denmark, Krogsgaard (Acta Pediat. **5**:103, 1926); Minneapolis, Harrington and Myers (Am. Rev. Tuberc. **14**:454, 1926); Philadelphia, Opie (J. A. M. A. **87**:1549 [Nov. 6] 1926); New York, Bellevue (from table 1); Dortmund, Sander (Deutsche med. Wchnschr. **47**:532, 1921); Ontario, Elliott (Tubercle **7**:122, 1925).

---

19. McLean, S., and Jeidell, H.: Data in 3,742 Pirquet Tests, Am. J. Dis. Child. **24**:73 (July) 1922.

20. Wahl, S. A., and Gerstenberger, H. J.: A Statistical Study of Tuberculin Tests in Infancy and Early Childhood, Arch. Pediat. **40**:143, 1923.

21. Cattermole, J. H.: Tuberculosis in Childhood, Columbus M. J. **16**:244, 1919.

22. Freedman, E.: Results of Pirquet Test in 464 Colorado Children, Columbus M. J. **16**:246, 1919.

Slater [23] tested children in a farming community living widely separated and hence exposed to few persons outside their own families. He used the Pirquet test, and therefore it is fair to assume that this helps to account for the lowness of the positive percentages.

Therefore these American statistics merely show the incidence of the Pirquet test in those suspected of having tuberculosis or in those coming in contact with tuberculous persons, not the percentage of tuberculous infection in the general population. Figure 10 shows graphically the lack of correspondence in the American statistics.

A notable exception is the series of Elliott [6] (fig. 11) giving the result of tests on children from many different schools and communities in Ontario. He used the Mantoux (0.1 mg.) test, and his results correspond closely to those obtained at Bellevue. It should be noted that the two curves are almost identical.

Harrington and Myers,[24] in studies of 2,118 Minneapolis school children, found an incidence but slightly higher than that shown by the statistics at Bellevue, and the slope of the curve is almost identical.

Opie's [25] figures from Philadelphia correspond closely to the slope and height of the curves made from results from New York, Ontario and Minneapolis.

Recent series reported from Europe by Mantoux tests show curves which run closely parallel to that from Bellevue. In the Dortmund series (Sander,[26] 1921, quoted by Barchetti [27] the curve is identical, and in other places, Munich, 1919, Graz, and Denmark,[7] while the incidence of positive reactions is somewhat higher, the curves are almost parallel to our figures. This is an interesting fact and suggests the thought that given the amount of tuberculosis existing in any community, the child's chances of infection at any age can be estimated by the slope of these curves. Figure 11 shows the results from several of the important European and American cities. All are based on Mantoux tests except Hamburger's original curve (Stich test). It is noteworthy that the curves for the two Austria cities, Vienna and Graz, are distinctly higher than any of the others, and that all the curves for American cities show about the same incidence.

---

23. Slater, S. A.: Pirquet Tests in a Rural Community, Am. Rev. Tuberc. **10**:299, 1924.

24. Harrington, F. F., and Myers, J. A.: Tuberculous Infection among Minneapolis School Children, Am. Rev. Tuberc. **14**:454, 1926.

25. Opie, E. L., and McPhedran, F. M.: Spread of Tuberculosis within Families, J. A. M. A. **87**:1549 (Nov. 6) 1926.

26. Sander: Ueber die Verbreitung der okkulten Tuberculose unter den Kindern, Dortmund, Deutsche med. Wchnschr. **47**:532, 1921.

27. Barchetti, K.: Zur Haufigkeit Tuberculose in Kindesaltern, Arch. f. Kinderh. **71**:180, 1922.

It is evident that there are only about half as many children infected at each age in this country as in the first series published from Europe, and that not over from 40 to 50 per cent of American children in the great cities harbor the bacillus even at puberty. The class of people from which the patients at Bellevue are drawn is almost, if not quite, the worst in the city. They are poor Irish, Italian and Jewish people, with a few negroes, from the lower East side, living in the most crowded sections. The incidence of tuberculosis among this class is probably higher than among people in the less congested parts of the city.

It is a hopeful sign to find the percentage of positive reactions so low. It means that American children are not all infected by the time of puberty. It also makes the tuberculin reaction an important one.

The value of the tuberculin reaction is generally misunderstood by the medical profession. The impression exists that nearly all children are infected, and therefore a positive reaction means little or nothing. This idea is based on the widely promulgated early European statistics, most of which corresponded roughly to the figures for Vienna (Jeannerett [11]). But if the amount of infection is so much less in this country, then it is of the greatest importance that those children who are infected should be watched through childhood and guarded against the stress and strain of adolescent life when active or fatal tuberculosis is so apt to break out. It must be remembered that "the army of the tuberculous is recruited from those infected in childhood" and "phthisis is the last verse of a song the first of which was sung in the cradle." These infected children, then, who carry tubercle bacilli in their bronchial nodes or elsewhere, are the ones who need early, correct diagnosis. The Mantoux test provides the means of making it. It should be used on every child who is underweight, anemic or languid or who has an unexplained irregular fever. These children with "tuberculous infection" so called, have true incipient tuberculosis, latent or active. Their disease is generally in lymphatic tissue, where it is easily encapsulated and where scar tissue does no great harm. Under intelligent, watchful care they usually do well. When the disease has involved more important structures, such as the lungs, bones or meninges, the damage is greater, the cure is more difficult or impossible, and the diagnosis has been made too late.

135 East Sixty-Fifth Street.

THE SIZE OF THE REACTING AREA IN INTRACUTANEOUS TUBERCULIN TESTS IN RELATION TO CLASSIFICATION OF DISEASE AND TO OTHER CLINICAL FACTORS *

LLOYD B. DICKEY, M.D.
SAN FRANCISCO

Soon after the introduction of the cutaneous test of Pirquet and the intracutaneous test of Mantoux, it was noticed that persons varied greatly in skin sensitiveness to tuberculin, the active principle of which has been recently proved by Seibert [1] to be a true protein. Various reasons have been ascribed for this difference in the grade of skin reaction. It was at first naturally believed to be related to the degree of tuberculous activity, or to the amount of tuberculotoxins made by the living tubercle. Later, persons were noted to be hypersensitive to tuberculin in whom no active tuberculous processes could be demonstrated. Krause [2] and Willis [3] have both submitted experimental evidence that the degree of sensitization is related to the time of infection, and that it is increased by reinfection.

It is not my intention to discuss in detail the nature of the tuberculin reactions. In view of Seibert's work, it may be assumed, and is generally agreed, that the positive reactions obtained with artificial tuberculin are evidences of sensitiveness to protein, or true allergy, established some time after the initial infection with the tubercle bacillus. The present study was initiated in an attempt to determine whether any clinical significance could be attached to the size of the visibly reacting area, when single intracutaneous tests were performed under conditions similar to office practice, with tuberculin purchased on the open market.

---

* Submitted for publication, Aug. 5, 1929.

* From the Department of Pediatrics, Stanford University Medical School.

* Read before the Section on Diseases of Children at the Eightieth Annual Session of the American Medical Association, Portland, Ore., July 12, 1929.

1. Seibert, Florence B.: The Chemical Composition of the Active Principle of Tuberculin: X. The Isolation in Crystalline Form and Identification of the Active Principle of Tuberculin, Am. Rev. Tuberc. **17**:402 (April) 1928.

2. Krause, A. K.: Studies in Immunity to Tuberculosis: Experimental Studies on the Cutaneous Reaction to Tuberculoprotein: I. Factors Governing the Reaction, J. M. Research **35**:1 (Sept.) 1916.

3. Willis, H. S.: Studies on Immunity to Tuberculosis: The Waning of Cutaneous Hypersensitiveness to Tuberculin and the Relation of Tuberculo-Immunity to Tuberculo-Allergy, Am. Rev. Tuberc. **17**:240 (March) 1928.

The reactions studied in this series were those in positive tests obtained during a study launched by the San Francisco Tuberculosis Association on the incidence of tuberculous infection in children residing in the vicinity of San Francisco. New patients entering the Stanford Children's Clinic were tested with tuberculin without reference to their complaints. The intracutaneous method was chosen because the dosage could be more easily controlled; 0.1 cc. of a 1 : 1,000 solution of Koch's old tuberculin, or a dose of 0.1 mg., was used in all the tests in this series. The flexor surface of the left forearm was the site chosen for the intracutaneous injection in all the patients. The tuberculin used was all purchased from one biologic supply laboratory, and was all manufactured in the same way, from one or the other of three different laboratory strains which were relatively nonvirulent. The results of the tests were read after forty-eight hours, and only those were considered positive in which each diameter of the hyperemic area was 5 mm., or more.

Seven hundred positive reactions were obtained that were suitable for inclusion in this series. The resulting area of erythema approximated a circle in shape in many of the tests, and in the others was elliptical. The two diameters were measured, and the area roughly computed from these measurements by using the equation for determining the area of an ellipse, $\frac{\pi A \times B}{4}$, or $A \times B \times .785$. If the diameters of the reacting areas can be measured to within 1 mm., it is probable that the error is within 10 per cent.

The average size of the 700 reacting areas was 459 sq. mm. This figure closely approximates the area on the surface of a silver quarter dollar. The tests in 369 boys averaged 439 sq. mm. in size; in 331 girls, 481 sq. mm.

When the reactions were compared with the ages of the children, it was noted that the smallest reactions occurred in the first year group, and that there was an increase in the size of the reactions with each added year of age up to the sixth year (chart 1). None of the nine positive reactions in the first year group reached the general average in size; in the second year group, only four of the twenty-seven did.

The oriental races gave considerably larger reactions than other races, the latter all approximating the general average (chart 2).

In those children who gave a history of contact with cases of active tuberculosis in the home, the average size of the reactions was greater than in those for whom no history of contact could be obtained (chart 3). The reactions were less strong in children whose contact with the disease was through the mother than in those whose contact was through the father or some other member of the family. The greatest reactions of all were given by a small group of eight whose contact was double, both mother and father having open tuberculosis.

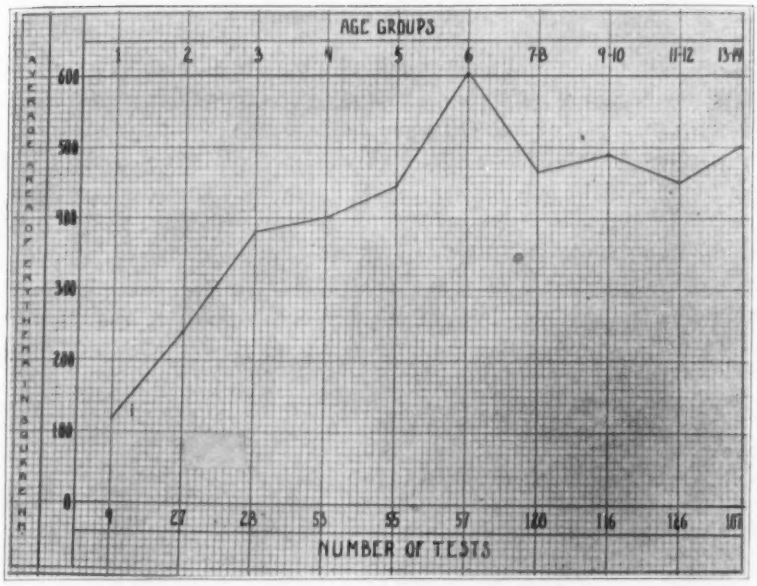


Chart 1.—The average size of the intracutaneous reactions to tuberculin in various age groups.

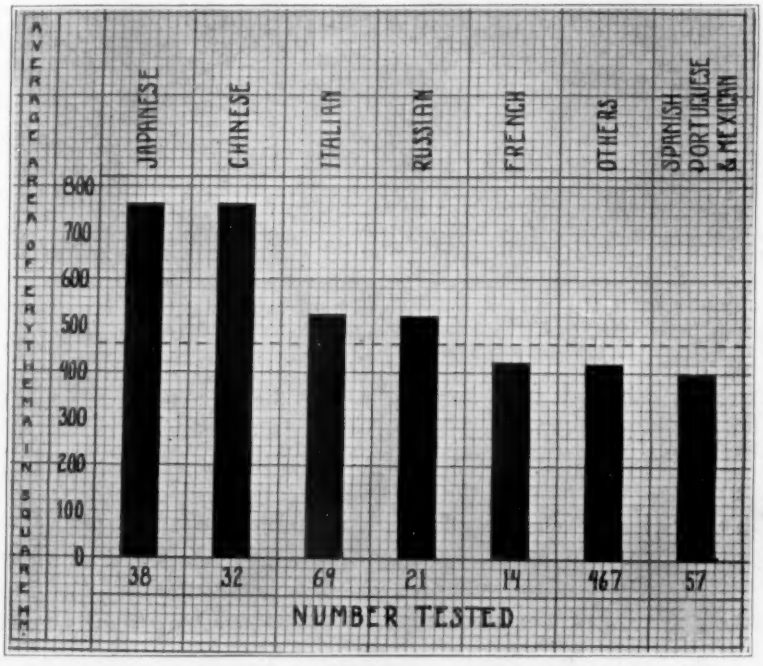


Chart 2.—The average size of the skin reactions in various racial groups. The dotted line is the height of the general average of all the 700 reactions.

Certain roentgenologic observations were considered. The reactions of the children (327) showing evidence of enlarged hilar nodes averaged 438 sq. mm. and did not vary greatly in grade from the reactions of those (346) in whom there was no evidence of enlarged hilar nodes, the reactions of the latter averaging 485 sq. mm. The reactions of those showing evidence of calcifications in the parenchyma (327) averaged 494 sq. mm., while the reactions of those without roentgenologic evidence of calcification (354) averaged 432 sq. mm. Those children in whom roentgenograms disclosed markings extending to the third zone (as described by Austrian and others [4]) had, on the average, larger

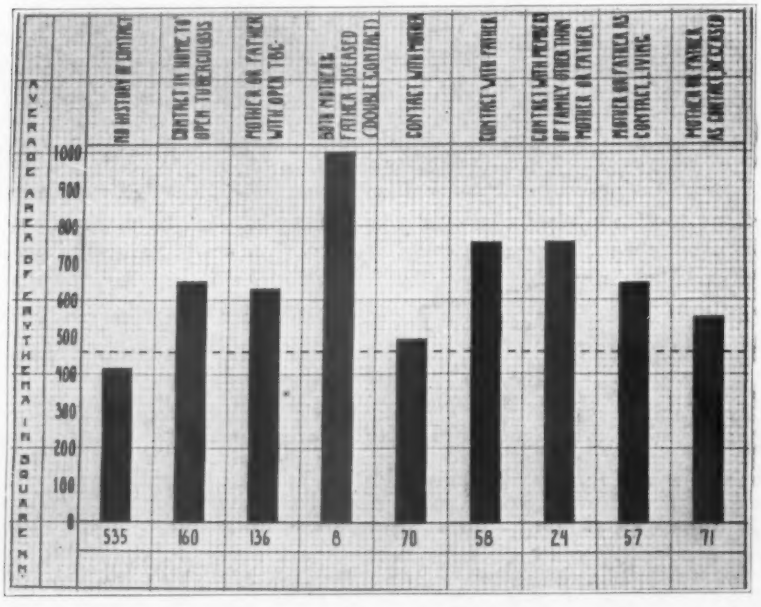


Chart 3.—The average degree of cutaneous sensitivity in children with no history of contact, and in various groups of those exposed to cases of open tuberculosis. The dotted line is the general average of all the 700 reactions.

tuberculin reactions than those in whom the markings were not so extensive (chart 4).

In children in whom active bone and joint disease, or cervical lymph node disease, was present, the reactions averaged greater than in those with active hilar disease, while the latter gave slightly larger reactions than those in whom no active disease of any kind could be demonstrated or was suspected, in the group classified as latent tuberculosis. Those giving evidence of the adult type of pulmonary tuberculosis reacted

4. Austrian, C. R., and others: Clinical and X-Ray Findings in the Chests of Normal Children, Am. Rev. Tuberc. **6**:331 (July) 1922.

less strongly to tuberculin than the other types (chart 5). Activity and latency were diagnosed in all types without reference to the size of the reaction to tuberculin. Those children in whom acid-fast bacilli were recovered from some diseased organ showed reactions lower than the average, as did also those who were known to have died later of tuberculosis. In patients who gave evidence of splenic enlargement, either on physical examination or by roentgenogram, the reactions were comparatively small.

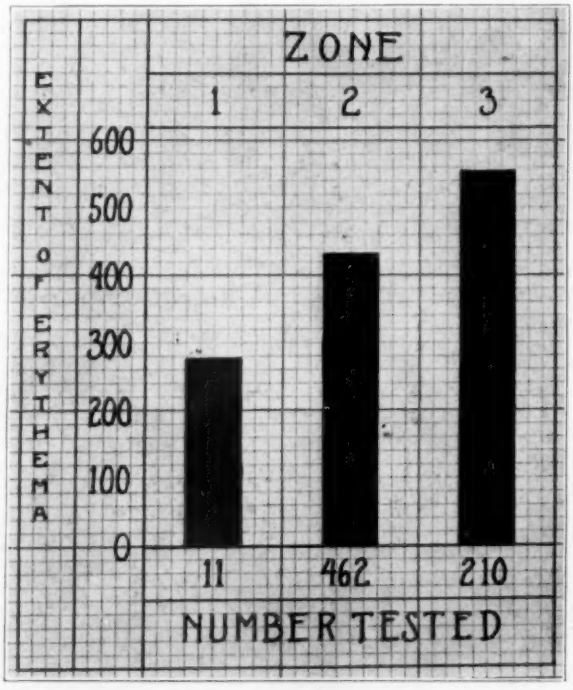


Chart 4.—The average size of the reactions in patients grouped according to the extent of the markings in roentgenograms of the chest.

Children with active phlyctenular conjunctivitis had reactions that averaged more than twice the size of the general average. Of twenty-nine patients with this condition, in only one did the skin test give a result below the average; in this one the reaction measured 400 sq. mm. With the exception of but three other tests in this group, all gave results of more than twice the average size.

The small group of four with erythema nodosum had reactions only slightly above the average in size (chart 6).

Those showing clinical evidence of bronchial spasm (asthma) had comparatively small reactions.

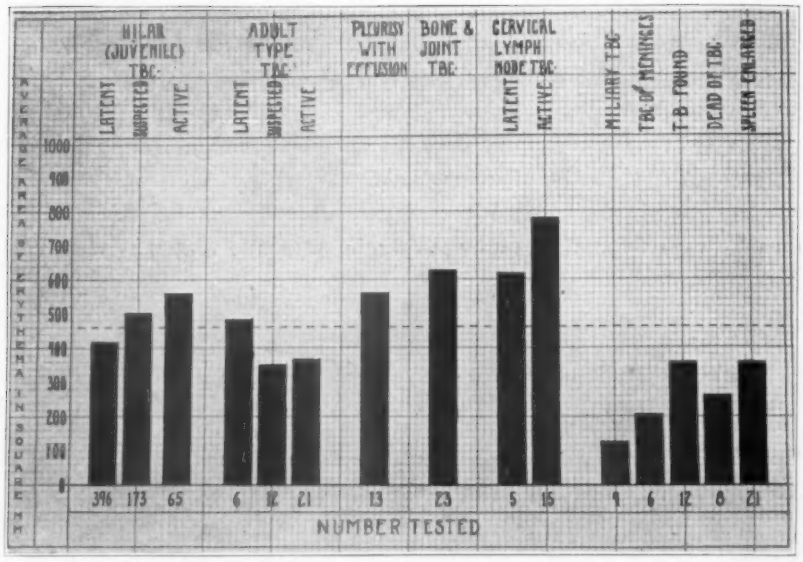


Chart 5.—The average degree of sensitivity in various types of tuberculous disease. The dotted line is the height of the general average of all the 700 reactions.

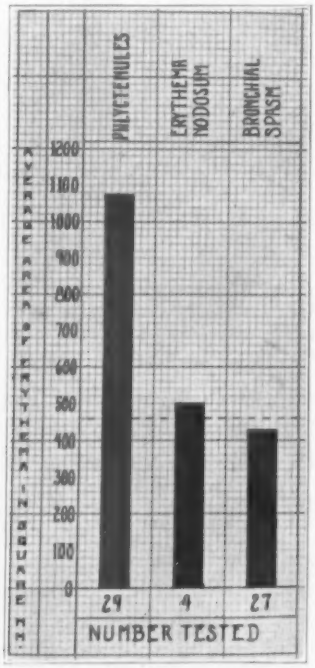


Chart 6.—The average size of the reactions in conditions sometimes associated with allergy.

COMMENT

Soon after skin testing in tuberculosis was first performed, White and Van Norman and others [5] noted that the same patients reacted to different dilutions of tuberculin with different intensity. No comparisons were made between individual reactions with the same dosage of tuberculin. Krause [6] and others have also noted the complete disappearance of sensitivity to tuberculin following the healing of tuberculous processes. The importance of repeated testing of tuberculous persons and a study of changes in sensitization such as has been made by Holmes [7] should be emphasized, but it is also important to know whether any significance can be attached to the intensity of single reactions. In the diagnosis, treatment and prognosis of tuberculosis in children, information must be gathered and evaluated in the shortest possible time.

If hypersensitiveness to tuberculin were in direct relation to activity of infection, one might expect no general difference in reactions in different age groups, such as occurred in this series. It is generally believed that tuberculous disease in infants and young children is more apt to be active than it is in older children and adults. The age incidence of the grade of skin reaction noted here might be interpreted as evidence in favor of the theory of the relation to the time of infection, as the maximal reactions tend to occur in older children, and the latter are, of course, more apt to be reinfected. But if hypersensitiveness means recent infection only, larger reactions should predominate in younger children; if it means reinfection, our curve is compatible and suggestive. Another factor that must be taken into consideration, however, before the age incidence is properly evaluated, is the relation between the size of the skin reaction and the total skin surface area. Though the actual size of the reaction in infants and younger children may be smaller, relatively it probably is not.

It is noted that cutaneous hypersensitiveness is marked among the children of oriental parentage, living under conditions in which adult

---

5. White, W. C., and Graham, D. A. L.: A Quantitative Modification of the von Pirquet Tuberculin Reaction and Its Value in Diagnosis and Prognosis, J. M. Research **20**:347 (April) 1909. White, W. C.; Graham, D. A. L., and Van Norman, K. H.: Studies of Tuberculin Reaction, with a New Index to Dosage in Tuberculin Treatment, Tr. Fifth Annual Meeting of the National Association for the Study and Prevention of Tuberculosis **5**:223, 1909. White, W. C., and Van Norman, K. H.: An Individual Quantitative Index to Tuberculin Dosage in Treatment, Arch. Int. Med. **6**:449 (Oct.) 1910.

6. Krause, A. K.: Human Resistance to Tuberculosis at Various Ages of Life, Am. Rev. Tuberc. **11**:303 (June) 1925.

7. Holmes, J. B.: Variation in Tuberculin Hypersensitiveness During the Course of Pulmonary Tuberculosis, Tr. National Association for the Study and Prevention of Tuberculosis **10**:133, 1914.

tuberculosis is rampant, and in which reinfection must constantly occur. The death rate for tuberculosis in the Chinese of San Francisco is over four times that for tuberculosis in the population at large,[8] and is probably about the same as that for tuberculosis in the Japanese. The incidence of phlyctenular conjunctivitis among children in this group is higher than that in children in general.

Children who are in contact with open cases of tuberculosis seem somewhat more sensitive to tuberculin than others, possibly because reinfection is constantly taking place.

Holmes[7] writes: "Cases of quiescent incipient pulmonary tuberculosis and arrested advanced pulmonary tuberculosis and dying cases may all present the same degree of hypersensitiveness on a given day." Although this statement might hold also for cases observed by me, the general averages in sensitivity of many cases of different types vary. Harmstorf,[9] in 1928, using different dilutions of old tuberculin, noted no difference in the degree of sensitivity of "scrofulous" children as compared with those suffering from hilar, and other forms of pulmonary tuberculosis. Cummins[10] recently recorded heightened reactions in juvenile tuberculosis as compared with forms of adult tuberculosis. In the series of cases of phlyctenular conjunctivitis in which the patients were treated by Casparis,[11] all reacted strongly, before treatment, to intracutaneous tuberculin. My observations in the present series confirm those of Cummins and of Casparis. A hypersensitiveness to tuberculin being now generally accepted as an etiologic factor in most cases of phlyctenular conjunctivitis, heightened skin reactions are to be expected when this condition occurs. Bronchial spasm being also generally considered to be an allergic phenomenon in many cases of asthma, it is conceivable that hypersensitiveness to tuberculin could be a causative factor. In my cases, however, I could not show such hypersensitiveness by skin testing, such as is readily shown in most cases of phlyctenular conjunctivitis.

In the patients with various types of overwhelming tuberculosis, meningeal, choroidal, miliary, and in those about to die of the disease whom I have studied, the reactions were minimal as compared with the reactions in other patients. Such phenomena are of common occurrence,

---

8. Report on Tuberculosis in San Francisco, California & West. Med. **26**:831 (June) 1927.

9. Harmstorf, Ernst: Quantitative Untersuchungen über die Tuberkulinempfindlichkeit beim Kinde, besonders bei Skofulose, Monatschr. f. Kinderh. **40**:270, 1928.

10. Cummins, S. L.: The Significance of Intradermal Tuberculin Tests, Brit. M. J. **1**:336 (Feb. 23) 1929.

11. Casparis, Horton: Phlyctenular Keratoconjunctivitis: Its Etiology and Treatment, Am. J. Dis. Child. **34**:779 (Nov.) 1927.

and have been long recognized. It is well known that in overwhelming tuberculosis the intracutaneous reaction to ordinary doses of tuberculin may even be negative.

SUMMARY

1. In a series of 700 positive reactions to tuberculin, the average area of the erythema was about 459 sq. mm.

2. The size of the skin reaction varied greatly in the groups studied, with the exception of that in very young children and that in children having phlyctenular conjunctivitis.

3. There was no marked difference between the average size of the reaction in girls and that in boys.

4. There was a gradual increase in the size of the reaction from the first to the sixth year. This may not mean an increase in the degree of sensitization.

5. Children of oriental parentage, subject to continual reinfection, were more sensitive than other racial groups studied.

6. Children who had had contact with tuberculous persons reacted more strongly than children who gave no history of contact with the disease.

7. A relationship between sensitivity and the extent of the markings in roentgenograms is suggested, but no relation between sensitivity and other roentgenologic observations was found.

8. Children suffering from phlyctenular conjunctivitis, lymph node and bone and joint tuberculosis reacted more strongly than those with hilar disease, while the latter gave slightly stronger reactions than children suffering from an adult type of pulmonary tuberculosis.

9. The reactions of patients classified as having "latent tuberculosis" averaged slightly less in size than the general average. The difference is not sufficient to relate the degree of sensitization to the degree of tuberculous activity.

ABSTRACT OF DISCUSSION

DR. ERNST WOLFF, San Francisco: Concerning the larger size of the reacting area, strong infection plays an important part. I remember a girl, aged 15 years, who presented nothing but a marked reaction in the tuberculin test, and on inquiry about the possibility of infection, her foster mother said she had no knowledge of any. Forty-eight hours later, in coming for reexamination, she told me that she had found out that the child was exposed seven years before and had been living for one year in a family with open tuberculosis. A marked reaction in the tuberculin test should always urge us to find any special condition of exposure or reinfection. Concerning the close relation of the degree of reaction in the tuberculin test to markings in the roentgenogram, I am a bit skeptical. The series of nearly 100 cases of nontuberculous children presented yesterday showed five cases with markings to zone 3 and thirty-seven cases with increased markings up to zone 2. The most important theoretical question is why do we have, in certain

tuberculous conditions, marked sensitization, and why, on the other hand, have we a rather small degree of sensitization in processes which seem to be related in their clinical and pathologic picture? I think that Ranke's theory assuming three stages of tuberculous infection is of value as a working hypothesis. In the first stage of primary infection, allergy is established and a medium strong reaction to tuberculin is obtained. In the second stage of dissemination there is a marked allergy. It is interesting to notice in Dr. Dickey's paper that the conditions which have to be considered as belonging to the second stage, such as tuberculosis of bones, that of the eyes and that of the serous membranes, give a marked reaction to tuberculin. The third stage, which closely relates to the tuberculosis of the lung in adults shows again a lessened degree of sensitization.

Dr. Horton R. Casparis, Nashville, Tenn.: I am much interested in Dr. Dickey's statement that, as a rule, those children with phlyctenular conjunctivitis reacted violently to tuberculin. That has been my experience in a rather large series of cases of phlyctenular conjunctivitis scattered over a period of six or seven years. In an attempt to explain this violent reaction, I became impressed with the fact that from most of those who have phlyctenular conjunctivitis a personal or family history of allergy could be obtained. The suggestion was therefore offered that these persons may have become hypersensitive to tuberculin, with phlyctenular conjunctivitis as a manifestation of this hypersensitiveness, just as other members of the group present hay-fever, asthma and so forth, as a manifestation of hypersensitiveness to dandruff, pollen and so forth. I should like to know whether or not Dr. Dickey has made any observations in this connection.

Dr. C. Ulysses Moore, Portland, Ore.: I should like to ask Dr. Dickey whether he has had any experience with the effects of vitamin D on calcification in tuberculosis. We are giving from 20 to 40 drops daily of viosterol, but the number of cases is as yet too small to allow the drawing of conclusions. Cod liver oil in rickets has never been satisfactory in this climate, whereas activated ergosterol in the form of viosterol is surprisingly beneficial. Will a potent solution of vitamin D stimulate calcification in tuberculosis, and if so, what dosage is advisable?

Dr. C. A. Aldrich, Winnetka, Ill.: In making intradermal salt solution tests, we have always felt uncertain about the results in babies less than a year old. The skin is finely woven and tense. When you force in as much as 0.1 cc., you are likely to force some of the solution underneath the skin into the subcutaneous tissues. I should like to know whether Dr. Dickey does not think that that might have something to do with the small reaction in babies.

Dr. Lloyd B. Dickey, San Francisco: We have not observed the point in which Dr. Moore was interested. I believe that Dr. Aldrich's point about the reaction in small children is well taken. It is possible, of course, that the testing in babies is not as accurate as it should be. We were somewhat at a loss to evaluate the curve we got. In the group of patients with phlyctenular conjunctivitis in which Dr. Casparis was interested, we frequently had cases of asthma. I am sorry I cannot tell you the exact percentage. I should think, without knowing exactly, the incidence of allergic reactions, in general, was probably higher in that group.

I again want to emphasize the importance of repeated testing in cases of tuberculosis. By testing patients repeatedly, we can get much more information, even about their sensitivity and degrees of sensitization. The reason we undertook this study was that we have been asked by the students and by the visiting physicians

on the ward whether we can place any significance at all on a single skin reaction. It is often impossible to have a patient under observation long enough to make repeated skin tests. That is another reason why this study was made. I was a little disappointed when I assembled the data. I expected they would show more than they did, and I thought the differences would be greater. Of course, we can learn more by observing the patient over a considerable period, testing him from time to time.

INTERPRETATION OF ROENTGENOGRAMS OF THE CHEST IN CHILDREN BASED ON OBSERVATIONS AT NECROPSY

II. TUBERCULOSIS *

JOHN A. BIGLER, M.D.
CHICAGO

This study was undertaken for the purpose of ascertaining just what pathologic changes were present in the lungs of children to account for shadows in the roentgenograms of the chest. A careful history of every patient was taken, with repeated physical examinations and the necessary laboratory work. Roentgenograms of the chest were made at intervals during life. If the patient died, films were taken of the chest post mortem, and finally films were made of the excised lungs in many cases.

In a previous paper,[1] general roentgenographic interpretation, the normal chest, and the hilar and linear markings were discussed. Here, only the observations on tuberculosis in children will be considered. No children with open tuberculosis are admitted to the hospital. This study covered a period of more than one year.

Because of the fact that tuberculosis in children is an aerogenous infection in at least 90 per cent [2] of the cases, the importance of roentgenographic examination of the lungs cannot be overestimated.

The basis used for diagnosis of tuberculous infection in adults cannot be used for that in children. Koch,[3] in 1891, wrote on what is now known as "Koch's phenomena." When he injected tubercle bacilli into healthy guinea-pigs, there occurred with the lesion at the site of injection an involvement of the lymph nodes draining that area. When

---

* Submitted for publication, May 17, 1929.

* From The Children's Memorial Hospital and the Otho S. A. Sprague Memorial Institute Laboratory.

* Read before The Chicago Pediatric Society as an Inaugural Thesis, March 19, 1929.

1. Bigler, J. A.: Interpretation of Roentgenograms of the Chest in Children Based on Observations at Necropsy: I. The Hilum Shadows and the Linear Markings, Am. J. Dis. Child. **38**:978 (Nov.) 1929.

2. Naegele, O.: Virchows Arch. f. path. Anat. **160**:426, 1900. Ghon, A.: Der Primäre Lungenherd bei der Tuberkulose der Kinder, Berlin and Vienna, Urban and Schwarzenberg, 1912. Opie, E. L.: The Focal Pulmonary Tuberculosis of Children and Adults, J. Exper. Med. **25**:855, 1917. Opie, E. L., and Anderson, H.: First Infection with Tuberculosis by Way of the Lungs, Am. Rev. Tuberc. **4**:629, 1921. Parrot, quoted by Ghon: Beitr. z. klin. d. Tuberk. **40**:87, 1919.

3. Koch: Deutsche med. Wchnschr. **17**:101, 1891.

tubercle bacilli were injected into guinea-pigs that had had a previous tuberculous infection, a chronic lesion developed at the site of injection, and there was no involvement of the regional lymph nodes. Essentially, this is the difference between the first infection in childhood and the secondary infection in adult life. Also, in the adult, infection most frequently is found in the apexes, but in children under 12 years of age, it is rarely present there.

PATHOLOGIC CLASSIFICATION

No attempt is made to offer a classification of the various forms of tuberculosis, but for simplification, it is described under several pathologic headings, namely; (1) the primary focus, (2) the tuberculous hilum, (3) miliary tuberculosis, (4) fulminating tuberculosis, (5) epituberculosis, (6) tuberculous bronchopneumonia and (7) calcification.

The Primary Focus.—In children, one looks for the primary pulmonary lesion and the involvement of the tracheobronchial lymph nodes in the hilum draining that portion of the lung. This simultaneous involvement of the hilar lymph nodes with the primary pulmonary focus has been named the "primary complex" of Ranke.[4] Pathologically, it has been described by many writers.[5]

Depending on the degree of pathologic change at the site of the primary focus and in the regional lymph nodes, shadows will be found whereby tuberculosis can be diagnosed from the roentgenograms.

In the majority of cases only one focus is found in the pulmonary tissue, but occasionally, there may be more than one. The age of the lesion plays a more important rôle in its size than the age of the patient. Adult lesions have a tendency to remain chronic, while childhood infections have a tendency toward fairly rapid healing or rapid termination by hematogenous dissemination. The focus in the lung is as frequent in the lower lobes as in the upper lobes. It starts as an exudative process which may vary in size from a small area several millimeters in diameter to the involvement of a whole lobe. It usually starts subpleurally, and as it progresses it goes through the stages of infiltration, caseation, possible cavity formation, encapsulation and finally calcification, although healing may take place in any stage without calcium deposits. As a result of this pathologic process, one may find shadows on the film denoting tuberculous bronchopneumonia, caseation, cavitation, fibrosis and calcification, or no shadows at all.

---

4. Ranke, K. E.: Primaräffekt, sekundäre und tertiäre Stadien der Lungentuberkulose, München. med. Wchnschr. **39**:2153, 1913.

5. Footnote 2 (fifth reference). Kreuss: De l'hérédité parasitaire de la tuberculeuse humaine, Paris, 1898. Opie, E. L.: The Relation of Apical Tuberculosis of Adults to the Focal Tuberculosis of Children, J. Exper. Med. **26**:263, 1917. Footnote 2 (second reference).

Early in this process, there is most often no evidence of the lesion on the films, especially if the amount of involvement is small. If the lesion is demonstrable on the film, it is seen as a soft shadow with irregular outlines merging more or less into normal lung tissue. It is represented on the same basis as bronchopneumonia, from which differentiation is usually impossible unless the shadow described is connected with a characteristic hilar shadow.

Even later, as the infiltration subsides and encapsulation and caseation take place, it is difficult to demonstrate the lesion on the film with ordinary technic. Unless it is from 1 to 1.5 cm. in diameter, is situated near the lateral wall of the chest and not behind the diaphragmatic

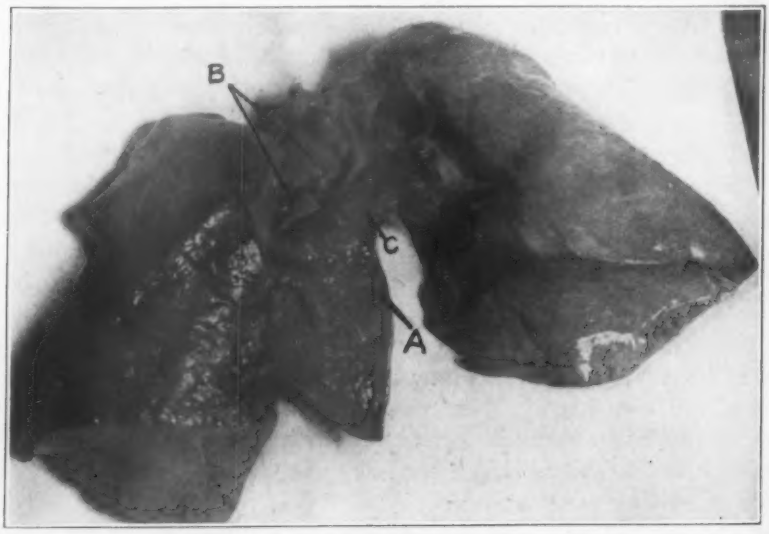


Fig. 1.—Lungs of child dying from a generalized miliary tuberculosis. *A*, caseated, encapsulated primary focus not visible on the chest film; *B*, caseated, hyperplastic hilum and paratracheal lymph nodes; *C*, carina lymph nodes.

shadow, is surrounded by a definite area of bronchopneumonia or atelectasis or contains calcium, it can rarely be demonstrated on the film. It has been found that caseation of a lesion does not mean that it will cast a shadow that is characteristic. I recall three cases in which pathologically a definite primary focus 1 cm. in diameter, walled off by a definite fibrous capsule and filled with a fairly dry caseous material, was found in the pulmonary tissue; yet in none of these cases could the lesion be demonstrated on any of the several films that had been taken of the chest (fig. 1).

As healing takes place, the lesion tends to become smaller. When calcium has been deposited, no difficulty has been experienced in dem-

onstrating the primary focus by the characteristic shadow that it casts (fig. 2).

It often happens that the shadow of the primary focus is incorporated in the shadow that extends out from the hilum on that side, and so in itself cannot be demonstrated (fig. 4). The reason

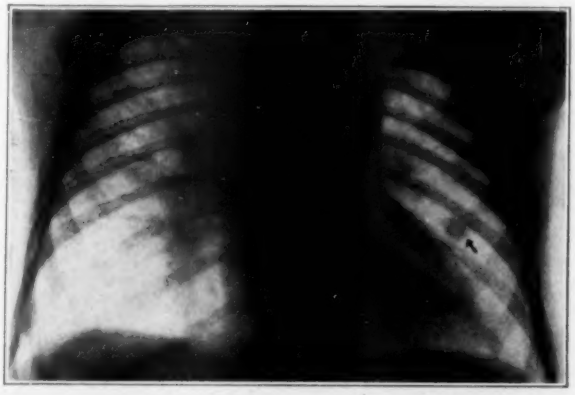


Fig. 2.—Calcified primary focus in left lower lobe. Wide mediastinal shadow due to thoracic Pott's disease and cold abscess.

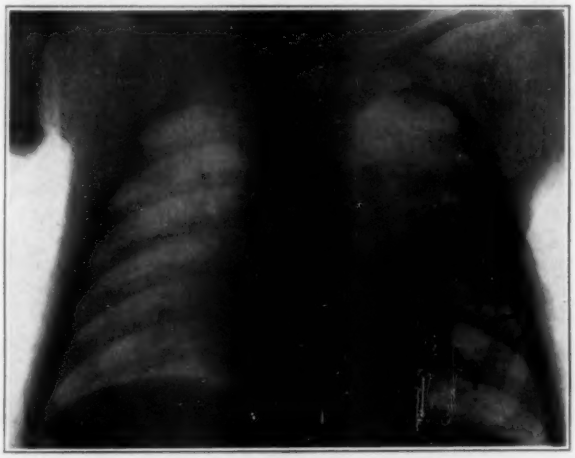


Fig. 3.—Massive tuberculous consolidation of left upper lobe with large cavity outlined by arrows. Miliary tuberculosis is also present, but is not discernible on the film.

for this is that the consolidation about the primary focus extends to the enlarged hilum, which gives rise to a shadow resembling a hilar or central pneumonia. Of eighteen cases of tuberculosis in which examination was made post mortem and in which there was no

evidence of calcification of the primary focus, only six allowed the location of the primary focus to be recognized on the roentgenogram, and in the majority of these six it was incorporated in the shadow from the hilum, which extended rather far out into the pulmonary tissue. In one case, the recognition was questionable.

Heavy trunk shadows occasionally are seen radiating into the hilum from a primary focus, but this is not the rule even in early cases. If found, they are an added bit of evidence of the specificity of the process, but it is not essential for the diagnosis.

The primary focus is found far more often in postmortem specimens than on the films. This is true not only because of the location of the focus behind denser structures in some cases or because shadows

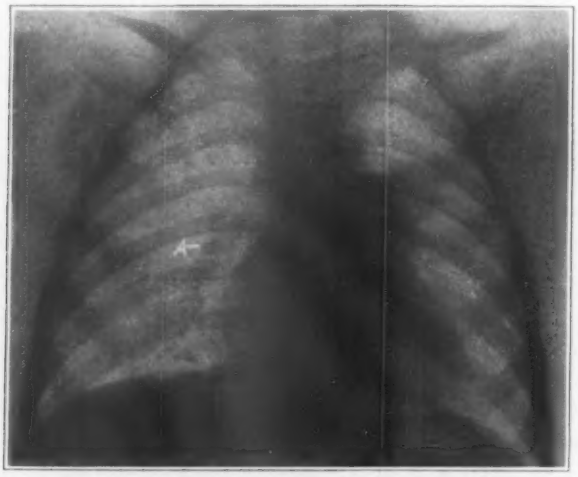


Fig. 4.—Typical triangular-shaped shadow extending from left hilum into left upper lobe due to tuberculosis. *A* shows a rounded shadow due to a blood vessel.

are not cast, but also because at times the primary focus may heal without leaving any noticeable evidence of its existence, as has been pointed out by several investigators.[6]

The primary focus may be recognizable without any evidence of involvement of the hilum, but absence of such involvement is far from the usual picture. When it does occur, the lesion is usually in the healed state when there has been calcification in the primary focus but none in the hilum.

---

6. Footnote 2 (second, third, fourth and fifth references). Krause, A. K.: The Healing and Disappearance of Tubercle, Am. Rev. Tuberc. **6**:238, 1922. Gardner, L. U.: Healing by Resolution in Experimental Pulmonary Tuberculosis, Am. Rev. Tuberc. **6**:163, 1922. Harms: Die Selbstheilung der Tuberkulose, Beitr. z. klin. d. Tuberk. **56**:318, 1923.

Tuberculous cavities were found pathologically on several occasions, but in only a few instances was it possible to demonstrate them in the roentgenogram (fig. 3). They were all small and were in areas of caseous bronchopneumonia, so that the contrast between the small cavity and the opaque shadow of the dense lung tissue was not great enough to make diagnosis a possibility.

The cavities were never large enough to demonstrate a fluid level, and they were aways surrounded by a definite area of increased density. In one instance, several small areas of decreased density not more than 1 cm. in diameter were found which represented a sacculated bronchiectasis in the region of the cavity. In another instance an area of decreased density was present in the enlarged hilar shadow,

Fig. 5.—Typical widening of right mediastinal shadow due to tuberculosis. The shadows of the miliary tubercles and the primary focus are not present.

and on postmortem examination it was found to represent a cavity in a large, caseous lymph node.

The Tuberculous Hilum.—I consider the hilar shadow important and have found it to be a more frequent guide to diagnosis from the film than the finding of a primary focus. The hilum is infected secondarily to the lung tissue, and it has been my experience that this involvement is usually more extensive, remains larger and leaves more recognizable scarring than the pulmonary infection. It is because of hilar shadows that many diagnoses of tuberculosis are made in error. Pathologic change in the hilum may be due to many different processes, some of which for a time resemble those due to tuberculous involvement, and it is here that history, physical and clinical manifestations and other laboratory data can be used to the greatest advantage.

The shadow of the tuberculous hilum can be best understood from the standpoint of its pathologic condition. It is made up of one or more of the following conditions: hyperplastic caseated lymph nodes with fibrous capsules; fibrous pleuritis with adhesions to the mediastinum, pericardium, wall of the chest or spinal column; pressure atelectasis and fibrosis of the surrounding lung tissue; consolidation of the surrounding lung tissue due to extending infection, and calcification or fibrosis of the lymph nodes. These pathologic conditions will give typical hilar shadows as described by Sluka[7] and Rach,[8]

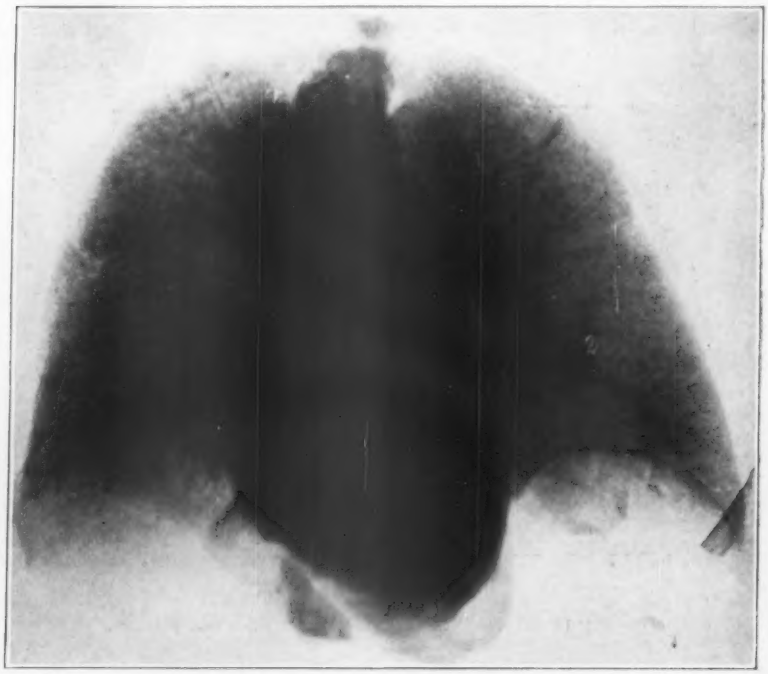


Fig. 6.—Lungs in case represented in figure 5. The right mediastinal shadow is made up of hilum and paratracheal caseated lymph nodes with a fibrinous pleuritis. Miliary tubercles are present, but no shadows are cast.

namely, triangular shadows with the base superimposed on the median shadow and with the apex reaching into the pulmonary field. This shadow may be homogeneous, but more often is of unequal density or may have a hazy appearance. It is fairly regular in outline and may present some arching as if pointing toward one lobe (fig. 4).

7. Sluka, E.: Further Contribution to the Diagnosis of Hilus Tuberculosis in X-Ray Plates, Wien. klin. Wchnschr. **26**:254, 1913.

8. Rach, E.: A Contribution to the Diagnosis of Pulmonary Tuberculosis in Children by Means of X-Ray, Ztschr. f. Kinderh. **8**:312, 1913.

If healing is taking place, it may contain areas of calcification and strandlike shadows. If there is involvement of the paratracheal lymph nodes, there will be an increase in the width of the mediastinal shadow on that side, which may be represented by a bulging shadow with a fairly definite outline or by a straight one with a fuzzy border (figs. 5 and 6). This is just as characteristic, when found, as the hilar shadow previously described, and often when healing takes place will contain calcified areas. A loss in the clear space between the right hilum and the right border of the heart is of importance in diagnosing pathologic change, as has been mentioned by Baum[9] (fig. 7). The shadow of the hilum is more easily seen on the right side, for on

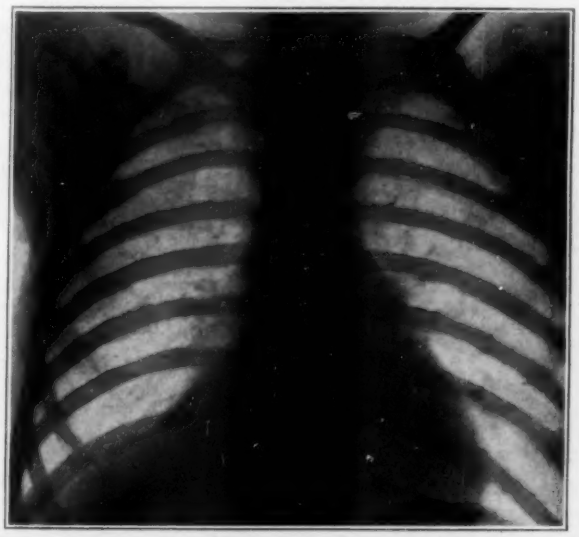


Fig. 7.—Tuberculosis of right lower lobe with typical hilar shadows and loss of clear space between right hilum and right heart border. This roentgenogram was taken while the patient was sitting.

the left side the heart shadow may occlude part of it. The hilar shadow may be represented by only a slight projection into the lung field, or it may extend out to a considerable degree, which is of importance in differentiating it from enlarged hila due to other causes. Even after extensive involvement, the hilar shadow gradually becomes smaller as healing takes place and calcification begins.

Miliary Tuberculosis.—In children, miliary tuberculosis is of frequent occurrence, and in all cases in which such a diagnosis has been

9. Baum, Felix: Roentgen-Ray Phenomena in the Normal and Pathologic Hilum, J. A. M. A. **88**:634 (Feb. 26) 1927.

made, the patients have died. In only about 50 per cent of the cases could the diagnosis of miliary tuberculosis be made from the roentgenograms (fig. 5). Often only comparatively few tubercles are present pathologically in the lungs. When the tubercles are caseated, numerous, from 2 to 3 mm. in diameter and surrounded by areas of exudation, then there is the characteristic snowflake appearance due to the merging of shadows from many planes in the lung on the one plane of the film, and the distribution is generalized to the very periphery (fig. 9). Even in films in which a miliary tuberculosis is readily seen, there may be no evidence of the primary focus or of an enlarged hilum.

Fig. 8.—Enlargement of right hilar shadow and shadows in right lung due to bronchopneumonia. Clear space still present between right hilum and right heart border. The arrows indicate areas of calcification. This roentgenogram was taken while the patient was sitting.

There may be partial healing of the primary focus or hilum, as evidenced by the presence of calcification. At times a widely disseminated bronchopneumonia will closely simulate miliary tuberculosis, and differentiation is possible only by the clearing of the former on successive films, by a distribution which is not so generalized to the periphery of the lung fields, by the absence of a positive tuberculin reaction and by the clinical appearance and course of the disease (fig. 10). It might be well to add that the Pirquet reaction was positive in 50 per cent

of the cases; this figure is intermediate between the practically 100 per cent of Hempleman [10] and the zero percentage of Levinson.[11]

Fulminating Tuberculosis.—In only one case was I able to observe pathologically a fulminating tuberculosis other than miliary. In this

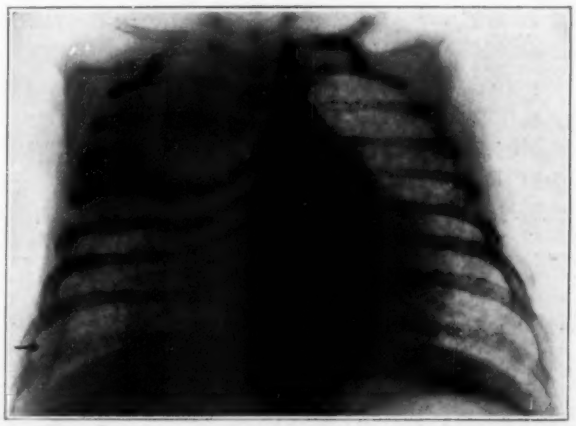


Fig. 9.—Typical shadows of miliary tuberculosis. Primary focus in the right upper lobe. The arrow indicates a shadow caused by fluid.

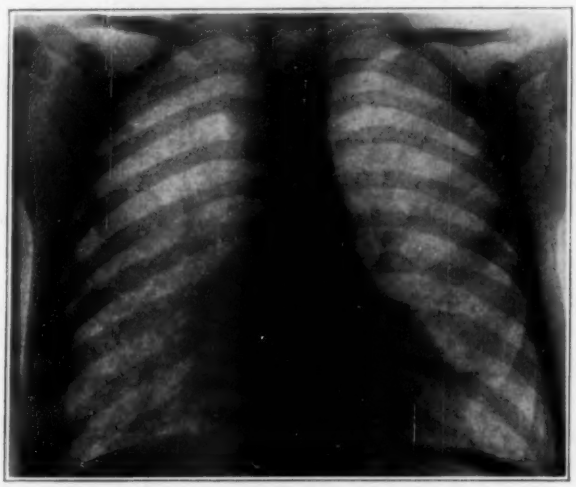


Fig. 10.—Shadows due to bronchopneumonia closely resembling those of miliary tuberculosis.

10. Hempleman, T. C.: Clinical Diagnosis of Pulmonary and Bronchial Gland Tuberculosis, J. A. M. A. **89**:1297 (Oct. 15) 1927.

11. Levinson, A.: Study of Active Tuberculosis in Children, Arch. Pediat. **45**: 390 (July) 1928.

child, the picture was complicated by a streptococcus pneumonia which involved all the lobes of the lung in a period of a few weeks. In several cases there was involvement of most of one lobe by a tuberculous pneumonic process which in itself would not have been recognizable on the film as a tuberculous process if it had not been accompanied by a typical hilar shadow on that side (fig. 11). In the case in which it is not accompanied by this hilar shadow, successive films following its course, the tuberculin reaction and the clinical course will lead to the correct diagnosis.

Epituberculosis.—Epituberculosis is a term used by Eliasberg and Neuland [12] and Kölzer [13] for a type of consolidation that is found in tuberculous children. It is attributed to a toxic reaction in the

Fig. 11.—Extensive tuberculous involvement of right upper lobe and right hilum. This roentgenogram was taken while the patient was lying on his back.

lung tissue surrounding a tuberculous hilum rather than to infection by the tubercle bacillus itself. The consolidation may involve one lobe or only a part of a lobe. It is difficult in a single film to distinguish epituberculosis from an ordinary pneumonia, but it can be diagnosed by successive films following its course, by the presence of a positive tuberculin reaction, by the occurrence of the condition in a patient known to have involvement of the hilum nodes and by the benign course of the disease over a variable period of weeks or months, with little temperature reaction and few clinical signs. Its spread is usually

12. Eliasberg, H., and Neuland, W.: Epituberculous Infiltration of the Lung in Children, Jahrb. f. Kinderh. **93**:88, 1920.

13. Kölzer, W.: Epituberculous Infiltration, Jahrb. f. Kinderh. **115**:194 (Feb.) 1927.

fairly rapid from the hilum to the periphery, and resolution always takes place fairly slowly from the periphery to the hilum. A similar process was described pathologically by Frankel [14] in 1895, while Sluka [15] wrote about the roentgen observations in a similar condition, but described it as due to direct infection with tubercle bacilli. Wessler,[16] in writing of an infiltration which begins at the hilum, used the term "recurrent hilus infiltration"; Greenberg [17] called it a "chronic tuberculous hilus pneumonia"; while St. Engel [18] thought it should be called a "paratuberculous infiltration." McPhedran [19] described an x-ray shadow which resembled this type of tuberculous infection.

Tuberculous Bronchopneumonia.—It has been possible to follow clinically the course of two patients who have a type of tuberculous infection which differs from the others described. One patient, a white girl, aged 10 years, has been followed for eighteen months, while the other, a Mexican boy, aged 2 years, has been followed for one year. The girl shows evidence of calcification in the pulmonary field and in the hilum. The boy has no evidence of calcification, but has Pott's disease of the lumbar region, for which he is being treated. The girl has involvement of only one side of the chest, while the boy has a bilateral involvement. On the films there is an extensive mottling, generalized from the hilum to the extreme periphery, with large discrete and conglomerate shadows, measuring generally 1 cm. in diameter. The same general character of the shadows has been present during the entire time they have been under observation (fig. 12). Both children appear in good general health, being fat and practically free from fever; both have markedly positive Pirquet reactions. Although I have made no postmortem examinations in this type of tuberculosis, I feel that it is a form of tuberculous bronchopneumonia which is chronic in its course, with little systemic reaction, and which clinically resembles epituberculosis. Although on the film there is a generalized distribution of the shadows, the condition is differentiated from acute miliary tuberculosis by the larger shadows, which may be unilateral, and by its chronic course over a period of months with benign systemic reaction.

---

14. Frankel, A.: Klinische und anatomische Mitteillungen über indurative Lungenentzündung, Deutsche med. Wchnschr., 1895, no. 10.

15. Sluka, E.: Ueber Roentgenbefunde bei Tuberkulosen Kindern mit expiratorischem Keuchen, Wien. klin. Wchnschr., 1910, vol. 32. Parrot (footnote 5, second reference).

16. Wessler, H., and Bass, M. H.: Recurrent Hilus Infiltration, Am. J. Dis. Child. **11**:198 (March) 1916.

17. Greenberg: Chronic Tuberculous Hilus Pneumonia in Children, Am. J. Dis. Child. **21**:65 (Jan.) 1921.

18. St. Engel: Ueber paratuberkulose Lungenerkrankungen, Berl. klin. Wchnschr. **42**:877 (Jan.) 1921.

19. McPhedran, F. M.: The X-Ray Diagnosis of Pulmonary Tuberculosis in Children, Am. Rev. Tuberc. **16**:420 (Oct.) 1927.

Pathologically, I encountered no cases of peribronchial or perivascular tuberculosis, although such a diagnosis was often referred to by the roentgenologist when he diagnosed as tuberculosis an increase of linear markings with beading and studding. If such a condition exists

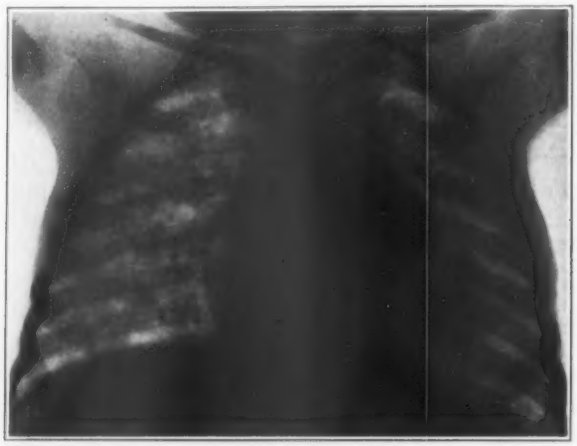


Fig. 12.—Chronic tuberculous bronchopneumonia. Compare with figures 9 and 10. This roentgenogram was taken while the patient was lying on his back.

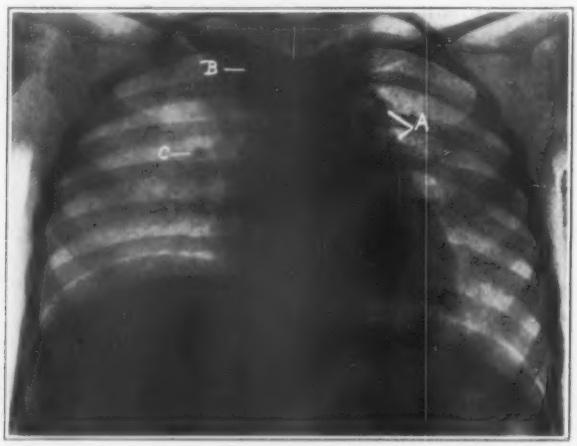


Fig. 13.—Healing tuberculosis. Calcification in *A*, left hilum and, *B*, left paratracheal lymph nodes; *C*, blood vessel.

pathologically in children, I do not see how it can be diagnosed from the roentgenogram.

Calcification.—Calcification is a term that is used frequently by all who interpret roentgenograms, and many diagnoses of tuberculosis have

been made on its supposed presence. Opie and McPhedran have said that the frequency of calcified nodes in the chests of children is overestimated, a statement with which I can readily agree. When the frequency with which they were diagnosed was checked up in this laboratory with their occurrence pathologically, it was realized how often error was being made. In looking over articles in the literature in which the frequency of calcification in roentgenograms was given with the incidence of positive tuberculin reactions, I found that in some instances the tuberculin reactions were positive in only from 40 to 50 per cent of the cases. This leads one to suppose that many of these

Fig. 14.—Excised lungs in case represented in figure 13. Calcifications in *1*, right paratracheal; *2*, carina; *3*, *4* and *5*, left hilum; *6* and *7*, intrapulmonary lymph nodes and in *8*, the remains of the primary focus.

so-called calcifications were densities due to some other cause, possibly vascular trunks. I grant that reactions to tuberculin can change from time to time,[20] for this has been my experience in a few cases. In only one of my cases showing calcification was the Pirquet test negative (two separate tests), but unfortunately a Mantoux test was not performed.

Opie [21] asserted that calcification in the lungs can be considered of tuberculous origin in practically all cases, because it so rarely occurs from other causes in children. In the small series of cases reported, calcification when present was always considered tuberculous because of its presence in a definitely tuberculous process. It has been stated that it

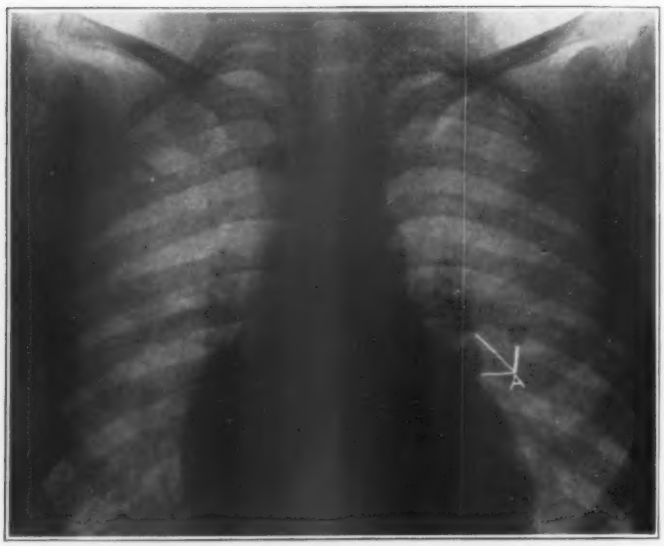


Fig. 15.—Typical mottled shadows of three areas of calcification (*A*). This roentgenogram was taken while the patient was sitting.

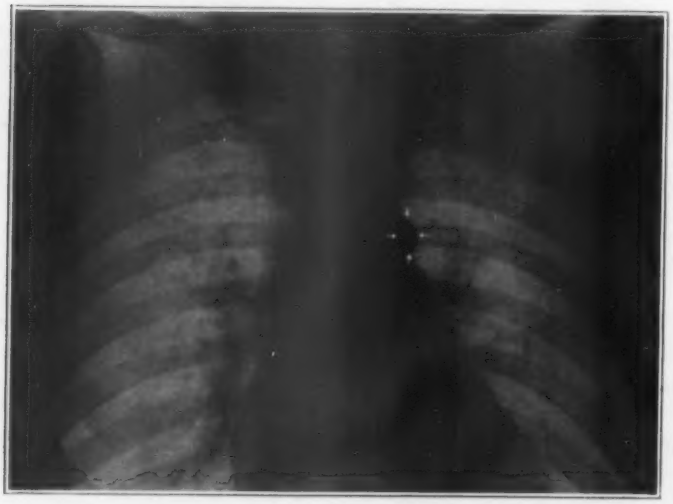


Fig. 16.—The arrows indicate a solid area of calcification in an older child. This roentgenogram was taken while the patient was lying on his back.

takes about three years for calcium to be deposited, but this is not always true, as I have found it twice in children under 2 years of age. It has also been reported in infants by Dunham.[22]

A calcified lesion casts a definite shadow. It is found comparatively rarely in infancy, but increases in frequency with age. It is an area of increased density usually irregular in outline, sharply demarcated from the surrounding tissue and visible even when beneath a rib. At an early age, it is rarely a solid shadow, but presents a speckled, flecked or lamenated appearance (figs. 13, 14, 15, 16 and 2). In children up to 9 or 10 years of age, it is most commonly not a perfectly round, circumscribed shadow of even density, but contains areas of unequal density in contrast to vascular trunks which are often multiple, larger near the hilum and smaller as they extend into the lung field, and which are of even density with fairly smooth margins that are not as sharply demarcated from the surrounding tissue.

Calcified lesions in children are more important than those in adults because they frequently represent only partial healing, from which a disseminated tuberculosis may originate. I cannot emphasize too strongly that more care must be taken in the diagnosis of calcification with its related diagnosis of tuberculosis. In 171 necropsies, the majority of which were performed on patients under 5 years of age, calcification was found in only five instances. Wollstein and Bartlett,[23] in reporting on 1,320 necropsies, found only eighteen instances of calcification, but 75 per cent of their patients were under 2 years of age.

CONCLUSIONS

Evidence of tuberculosis is not found pathologically as frequently as one would expect from the diagnoses of films of the chest. The incidence of tuberculosis in children admitted to the Children's Memorial Hospital is much lower than the figures quoted for tuberculosis in children.

---

20. Austrian, C. R.: Observations on Children Admitted to a Tuberculosis Dispensary, Tubercle **6**:29, 1924; The Evolution of Tuberculous Infection in Childhood, Am. Rev. Tuberc. **16**:501 (Oct.) 1927. Krause, A. K.: Human Resistance to Tuberculosis at Various Ages of Life, Tubercle **11**:303 (June) 1925. Hutchinson, D., and Myers, J. A.: Studies on Tuberculosis in Infancy and Childhood, Am. Rev. Tuberc. **14**:437 (Oct.) 1926.

21. Opie, E. L.: Pathological Evidence of First Infection in Association with Active Pulmonary Tuberculosis, Am. Rev. Tuberc. **10**:249 (Nov.) 1924.

22. Dunham, E. C., and Smythe, A. M.: Tuberculosis of the Cervical Lymph Nodes in Infancy, Am. J. Dis. Child. **34**:962 (Dec.) 1927.

23. Wollstein, Martha, and Bartlett, F. H.: A Study of Tuberculous Lesions in Infants and Young Children, Based on Postmortem Examinations, Am. J. Dis. Child. **8**:362 (Oct.) 1914.

The primary focus is found more often pathologically than on the x-ray film unless calcification is present. The more or less characteristic hilar, or mediastinal shadow, is the most frequent guide to the diagnosis.

Miliary tuberculosis can be recognized on the films in only about 50 per cent of the cases. It can be differenitated from chronic tuberculous bronchopneumonia by the smaller size of the shadows.

Repeated tuberculin tests when negative are important in ruling out a diagnosis of tuberculosis.

707 Fullerton Avenue.

CARDIOSPASM IN THE NEW-BORN INFANT

REPORT OF THREE CASES *

T. L. BIRNBERG, M.D.
ST. PAUL

Severe vomiting in the new-born infant is usually of grave portent, suggestive of a serious underlying pathologic process. The recognition of definite cardiospasm in three such cases has prompted an investigation of this unusual condition, as its detection in the new-born infant has hitherto not elicited comment.

Cardiospasm is defined as a spasm of the smooth muscle fibers surrounding the cardiac orifice, resulting in partial or complete occlusion of the lower end of the esophagus. It is therefore a functional disturbance of neurotic origin, causing failure of the lower end of the esophagus to consummate the act of deglutition.

The earliest description of cardiospasm was given by von Ziemssen and Zenker in 1824. In 1833 Hannay applied the name "idiopathic dilatation of the oesophagus" to the condition, but in 1882 Mikulicz considered that the obstruction was due to simple spasm at the cardiac orifice; he applied to the spasm the now accepted term of "cardiospasm." In 1896, Rolleston demonstrated specimens of esophageal obstruction apparently due to the failure of relaxation of the ring muscle at the cardia. The early observations were made almost entirely on adults, and the condition has been recognized in infancy and childhood only in recent years.

The occurrence of cardiospasm in infancy has been observed infrequently, the literature containing but few references to this condition in children. Diagnosis of the condition in the new-born infant has not been recorded, although many of the patients observed in the later months of infancy and childhood give a definite history pointing to cardiospasm existing at birth. In 1910, Beck [1] reported two cases in infants, aged 2 and 3 months, who vomited milk immediately after ingestion, unmixed with gastric juice, while milk introduced directly into the stomach was never vomited. In 1908, Göppert [2] reported a similar case of an infant, aged 8 months, who recovered at the ninth

* Submitted for publication, Sept. 21, 1928.

* Read before the Section on Diseases of Children at the Seventy-Ninth Annual Session of the American Medical Association, Minneapolis, June 14, 1928.

1. Beck, C.: Kardiospasmus in Säuglingsalter, Monatschr. f. Kinderh. **9**:555, 1910.

2. Göppert: Der Kardiospasm, Therap. Monatschr. **22**:390, 1909.

month. In 1911, Morgan [3] cited the case of an infant, aged 9 months, who began to vomit at the age of 3 weeks, and who became progressively worse. Relief was obtained only by the passage of a catheter through the cardia, although it was inserted with difficulty, and by feeding through this catheter, which was left in place for varying periods until the cardia finally remained permanently open. Morgan also quoted the record of Mery's case of cardiospasm in an infant, aged 10 months, who had vomited often since birth. In 1914, Laroziere reported the case of an infant, aged 16 months.

Langmead [4] cited a typical case in an infant who from the time of the first feeding had vomited often. X-ray examination showed retention of most of the meal in the dilated lower part of the esophagus for three hours. A stomach tube could not be passed, but the esophagoscope passed freely into the stomach, revealing no organic obstruction.

Hess [5] observed eight cases of abnormal irritability and spasm of the pharynx, cardia and pylorus in 100 new-born infants in whom he was determining the size of the normal pylorus by the passage of catheters. These infants demonstrated spasm at these points sufficient to interfere with the passage of the catheter. A definite history of severe vomiting which continued through early infancy was obtained in two of this group. Hess also reported cases of cardiospasm in which the condition interfered with the passage of a catheter into the stomach in one small infant, aged 7 weeks, and in two infants, aged 3 months. All of these gave a history of frequent vomiting. A report of the x-ray observations, treatment or final outcome was not given. He quoted a fourth case of explosive vomiting in an infant, aged 3 months, showing cardiospasm and pylorospasm, accompanying congenital obliteration of the bile ducts. At autopsy, pathologic changes were not found in the stomach. Hess suggested that cardiospasm is far more frequent in infants than is generally recognized.

Bruce [6] reported the case of a child, aged 3 years who had had intermittent attacks of choking since birth. X-ray examination showed spasm of the cardia occurring after the first portion of the meal entered the stomach, therefore causing complete occlusion; choking occurred when food flowed over from the filled esophagus into the larynx. The administration of atropine was ineffective, but benzyl benzoate in large doses produced relaxation of the spasm.

---

3. Morgan, W. G.: Cardiospasm in Infants, M. Rec. **80**:173, 1911.

4. Langmead, F.: Esophagectasis in an Infant, Proc. Roy. Soc. Med. **13**:43, 1920.

5. Hess, Alfred: The Pylorus, Pylorospasm and Allied Spasms in Infants, Am. J. Dis. Child. **7**:184 (March) 1914.

6. Bruce, J. W.: Cardiospasm in a 3-Year-Old Child, Kentucky M. J. **19**:824 (Dec.) 1921.

Seeger [7] cited the case of a boy, aged 12 years. Messeloff, Shulman and Buckstein [8] reported the condition of cardiospasm in a girl, aged 11 years, which dated back one year. Caille [9] described the case of a boy, aged 10 years. Castronovo [10] recorded the case of a boy, aged 5 years, whose vomiting began at birth.

Freund [11] cited the case of an infant, aged 2½ years, whose condition was apparently due to a definite hysteria.

The standard textbooks on pediatrics and infant feeding throw little light on this subject. Abt [12] commented on the rarity of cardiospasm in infancy and childhood, while Holt, as well as Pfaundler and Schlossman made no reference to it.

Feer,[13] on the contrary, stated that the condition is not uncommon in infancy and childhood.

Kerley and LeWald [14] stated that cardiospasm may be congenital, but is extremely rare before the tenth year. They quoted the cases of a girl, aged 12 years, and a boy, aged 14 years, in both of whom the condition was of recent onset.

ANATOMY AND PHYSIOLOGY OF THE CARDIA

The existence of so definite an entity as cardiospasm would suggest that a well defined cardiac sphincter must exist normally, and especially in the cases of spasm. Strangely enough, this is not the case. Some authorities doubt whether a cardiac sphincter exists. Section of the lower end of the esophagus shows a thickened muscular coat, possessing sufficient muscle tone to offer a definite resistance to the passage of foods at the cardia. The muscle thickening does not correspond in strength to the sphincters at the pylorus, ileocecal valve and anus, its main function being apparently concerned more with the prevention of regurgitation of gas and fluid upward and only slightly with the regulation of the onward passage of the esophageal contents.

As food is propelled down the esophagus by the peristaltic waves, its progress is arrested by the cardia for five or six seconds, which allows only a thin threadlike stream to pass into the stomach, and

7. Seeger, S. J.: Cardiospasm in a Boy of 12, Wisconsin M. J. **26**:154 (March) 1927.

8. Messeloff, C. R.; Shulman, H. I., and Buckstein, J.: Cardiospasm in Infancy and in Childhood, Am. J. Dis. Child. **35**:427 (March) 1928.

9. Caille, quoted by Messeloff, Shulman and Buckstein (footnote 8).

10. Castronovo, quoted by Messeloff, Shulman and Buckstein (footnote 8).

11. Freund, W.: Kardiospasmus, Monatschr. f. Kinderh. **2**:15 (Aug.) 1903.

12. Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1924.

13. Feer: Diagnosis of Children's Diseases, Philadelphia, J. B. Lippincott Company, 1925.

14. Kerley and LeWald: Digestive Disturbances in Infancy and Childhood, New York, Paul B. Hoeber, 1923.

which then promptly relaxes, so that the esophagus should be entirely empty within from five to seven seconds. Fluids and semisolids traverse the esophagus faster than solids. At the completion of the meal, the cardia remains closed and prevents the regurgitation of food and gas. In infants, however, the normal tonus is much weaker, as is evidenced by the frequency with which regurgitation occurs. This being the case, the occurrence of active spasm at the cardia in infancy is the more remarkable.

The activity of the cardia is regulated by counterbalanced nervous systems. The splanchnic nerves are inhibitory, and when stimulated, as by swallowing, produce a cessation of motion, and thereby an opening of the cardia. The vagus nerve supplies the motor function and causes contraction of the cardia.

ETIOLOGY

The cause of this condition has been responsible for speculation by many workers, and a variety of theories have been presented. Cameron,[15] whose monograph on vomiting in infancy is a classic, believed that temporary failure of the inhibitory apparatus of the bowel is apparently due to the tendency on the part of the inhibitory or splanchnic nerves to lag behind the motor or augmentary apparatus. The inhibitory activity of the skeletal musculature is similarly retarded at birth, as is suggested by the rigidity and spasticity of the new-born infant. This functional retardation of the inhibition is temporary and of varying degree. Pylorospasm exists in those infants in whom the condition is abnormally developed and prolonged at the pylorus. The failure of inhibition is most evident at the various sphincters, notably the pylorus and anal sphincters, to a lesser extent at the cardia, and the ileocecal valves. Esophageal obstruction, apparently due to the failure of relaxation of the ring muscle at the cardia, was demonstrated in specimens by Rolleston, in 1896.

The cardiac sphincter seldom undergoes any appreciable hypertrophy, because the pressure within the esophagus is low. Dilatation of the esophagus rather than hypertrophy is more likely to occur, in contrast to the stomach, with pylorospasm. In adults, cardiospasm is apparently due to the failure of the sphincter to relax, rather than actual spasm. The esophagus may become enormously dilated without showing any obstruction at the cardia or hypertrophy of the cardiac sphincter. The condition in adults is largely due to disease of the nerves of the esophagus, corresponding to the pathologic changes existing in heart block. Cases are also described as having resulted from tabes, diphtheria, or from reflex abdominal disorders, as duodenal ulcer, etc.

---

15. Cameron, H. C.: Vomiting in Infancy, Brit. M. J. **1**:765 (April 25) 1925.

Adults almost never give a history of symptoms dating back to infancy, but rather of development after maturity.

Walton [16] reported most cases as occurring in persons beyond middle age. In these cases he considered that there was a true spasm of the cardia, often sufficient to prevent the passage of the stomach tube, bougies or mercury tubes. The cardia grasps the bougie firmly when passed through, requiring definite effort to withdraw it.

Both Douglas and Morrison blocked phrenic nerves with alcohol in an effort to relieve cardiospasm, but without result, suggesting that the diaphragm is not responsible for the pathologic condition.

Hurst [17] coined the name "achalasia," or failure of the sphincter to relax. He described the dilatation of the esophagus which occurs in adults, and later develops hypertrophy of the walls above the obstruction, even though an actual hypertrophy of the ring muscle may not exist. A few autopsies on record show coincident hypertrophy of the pylorus and lower end of the esophagus.

Mutch [18] considered the condition to be the result of a defect in the neuromuscular mechanism at the lower end of the esophagus, capable of causing stasis of food, although a large bougie or finger can be pushed through the cardiac orifice without difficulty.

Jackson [19] suggested that many cases are due to the failure of the diaphragmatic pinchcock to open. This is the normal mechanism by which, in conjunction with the kinking of the esophagus below the diaphragm, regurgitation of the food in the stomach is prevented. The crura of the diaphragm act as a sphincter around the esophagus.

Shaw and Woo [20] ascribed the cause to an increase in the normal bend to the left of the esophagus as it pierces the diaphragm because of distention of the stomach, which thus causes obstruction of the esophagus.

Wilms [21] considered that spasm of the sphincters at the cardia, pylorus, anus and bladder are related and are not uncommon in infancy. Apparently, normal reflex stimuli are not sufficient to open these sphincters adequately. Thus, in cardiospasm, the normal swallowing reflex does not cause the cardia to open; but when the stronger reflex of vomiting acts, the cardia opens somewhat, thus permitting some of the food to enter the stomach.

---

16. Walton, A. J.: Surgical Treatment of Cardiospasm, Brit. J. Surg., vol. 12.
17. Hurst, A. F.: Sphincters of the Alimentary Canal, Brit. M. J. **1**:145 (Jan. 24) 1925.
18. Mutch, N.: The Treatment of Cardiospasm, Practitioner **107**:339, 1921.
19. Jackson, C.: Diaphragmatic Pinchcock in So-Called Cardiospasm, Laryngoscope **32**:139, 1922.
20. Shaw and Woo: Six Cases of Esophagetasia, Proc. Roy Soc. Med. **10**:1, 1917.
21. Wilms: Dauerspasmus, Deutsche. Ztschr. f. Chir. **144**:765, 1918

Held and Gross [22] classified the causes as follows: (1) inherited unstable vegetative nervous system, which may cause from childhood functional disorders of most organs supplied by the vegetative nervous system, as enuresis, periodic vomiting, laryngospasm, urticaria, etc.; (2) inborn status asthenicus or status thymicolymphaticus, resulting in hyperirritability of the vagus and sympathetic nerves, often causing functional disorders of the vegetative nervous system; (3) reflex irritation from diseased organs, as gastric ulcer, gallstones, etc.; (4) infections by toxic and metabolic agents, as nicotine, lead, uremia, etc.; (5) local esophageal disease, as fissure, erosions, ulcers, etc.

Plummer,[23] reporting the cases of forty adults, did not find any history of vomiting in infancy. He quoted the following theories as to causation: (1) primary cardiospasm (Meltzer); (2) primary atony of the musculature of the esophagus (Rosenheim); (3) simultaneous presence of cardiospasm and paralysis of the circular fibers of the esophagus, due to paralysis of the vagus (Kraus); (4) congenital disposition (Fleiner, Zenker, Huschka and Sievers); (5) primary esophagitis (Martin); (6) kinking of the hiatus of the esophagus.

Kelly [24] demonstrated experimentally that stimulation of the peripheral ends of a cut vagus nerve causes dilatation of the cardia. If the cut vagus is not stimulated, the cardia contracts, producing a condition analogous to cardiospasm. He suggested that hyperacidity may reflexly cause spasm of the cardia.

Diggle [25] reported a case occurring after influenza, while Rolleston recorded a case in a boy after pertussis.

REPORT OF CASES

CASE 1.—*History.*—The birth of K., a boy, had been normal, the weight at birth being 5 pounds 15½ ounces (2.6 Kg.). He vomited after the first feeding, and during and immediately after other feedings; at times the vomiting was projectile. Vomiting did not occur between feedings. He nursed from the breast at first, but was given breast milk by bottle on the fourth day, due to failure to nurse. He lost almost 1 pound (0.5 Kg.) in the first four days. The temperature was 101.8 F. on the fourth day; otherwise it was normal. Tube feeding of breast milk, 1½ ounces, six times a day, was then begun, with scarcely any vomiting thereafter. He began to gain weight at once. Two days later, tube feeding was stopped and thereafter he took bottle feeding. Difficulty in passing a catheter was observed only once. After the fifth day on bottle feeding, breast feeding was resumed, and the child vomited about once a day; vomiting continued in small amounts, not projectile, once or twice a day until he was weaned, at the age of 9 months. Growth had been normal since that time with no vomiting.

---

22. Held, I. W., and Gross, M. H.: Cardiospasm, J. A. M. A. **66**:233 (Jan. 22) 1916.

23. Plummer, H. S.: Cardiospasm, J. A. M. A. **51**:549 (Aug. 15) 1908.

24. Kelly, A. B.: Med. J., Oct. 19, 1912.

25. Diggle, G. H.: Cardiospasm, Practitioner, vol. 116, p. 304.

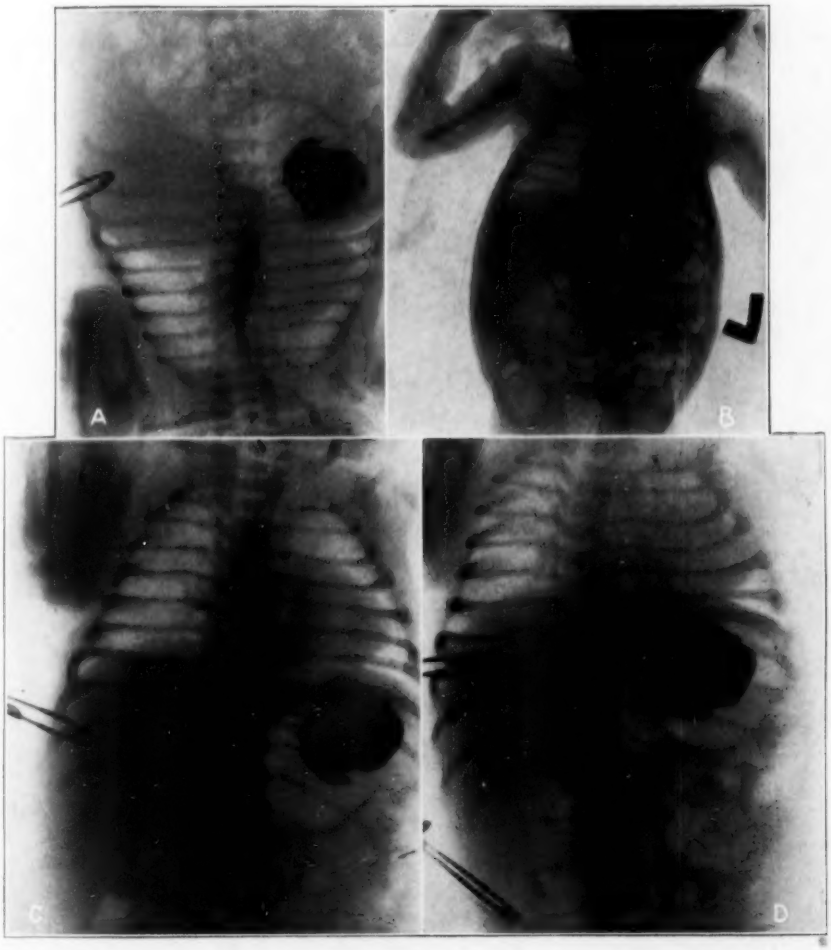


Fig. 1 (case 1).—*A*, roentgenogram taken on the fourteenth day of life. It took one and a half minutes until the cardia opened and the barium began to go into the stomach in a thin stream. The film was taken about from four to six minutes after the start of feeding. It shows a spindle-shaped shadow in the esophagus which is just emptied into the stomach. *B*, taken about five minutes later, from nine to eleven minutes after eating (some more feeding had been given). Note the complete occlusion of the cardia. The food mass in stomach is smaller; part of it is already in the small intestines. *C*, taken on the eighth day of life, immediately after feeding; it shows only slight delay (result of tube feeding). *D*, taken on eighth day; ten minutes after feeding shows no barium in the esophagus (result of tube feeding).

Examination.—On the fourth day of life, x-ray examination showed that the food stopped at the cardia; after from one to one and a half minutes, the food began to pass through in a fine stream. Peristaltic waves were visible in the esophagus. Barium was visible in the duodenum in five minutes. After the third day of tube feeding, the retention in the esophagus was much less, but pyloric obstruction was present as the barium did not leave the stomach during the first two hours; after six hours, some barium was still present in the stomach, showing that a moderate degree of pylorospasm had also developed.

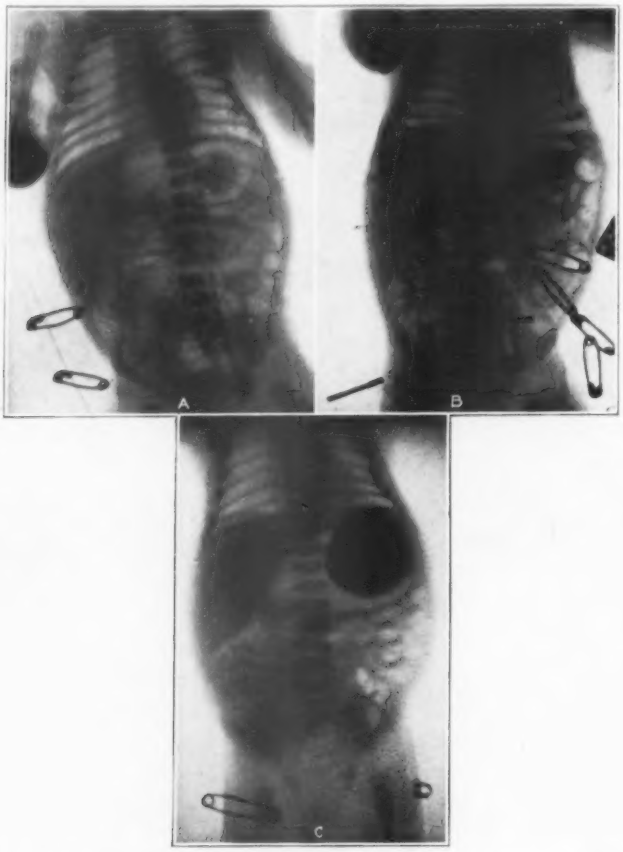


Fig. 2 (case 2).—*A*, taken on the fourth day of life, shortly after feedings; shows rather diffused filling of esophagus, with slow passage through the cardia (retention of one hour in the esophagus). *B*, taken on the sixth day of life; retention of barium in the esophagus, one hour and twenty-five minutes after the meal. No food has passed the pylorus. *C*, taken on the thirteenth day of life; it shows the esophagus completely emptied ten minutes after the meal. Barium passed through pylorus in one-half hour with normal emptying time.

CASE 2.—*History.*—The birth of S., a boy, had been normal, the weight at birth being 7 pounds 4 ounces (3.3 Kg.). Small amounts of brownish fluid were vomited during the first two days. On the third night, he vomited almost every feeding,

also water. He lost 1 pound 1 ounce (0.53 Kg.) in the first three days; the temperature was 102.5 F. on the third day. The vomiting was forcible through the mouth and nose; the infant seemed in pain until the emesis was complete. He was startled by slight noises. He seemed extremely hungry after vomiting.

Tube feedings were begun on the fourth day, with apparently slight resistance to the passage of a tube on the first attempt only. Emesis occurred after only one tube feeding. On the fifth day, emesis occurred three times, once forcible, apparently before the tube entered the stomach. The temperature was normal on the fifth day.

The child was given breast milk by tube only, until the tenth day; vomiting occurred in varying amounts about three times a day at this time. The gain in weight was steady. The temperature remained normal. The feeding by tube was gradually discontinued during the next few days and breast feeding was resumed with much less vomiting thereafter. After leaving the hospital on the sixteenth day, he vomited very little, and progress was normal in all respects.

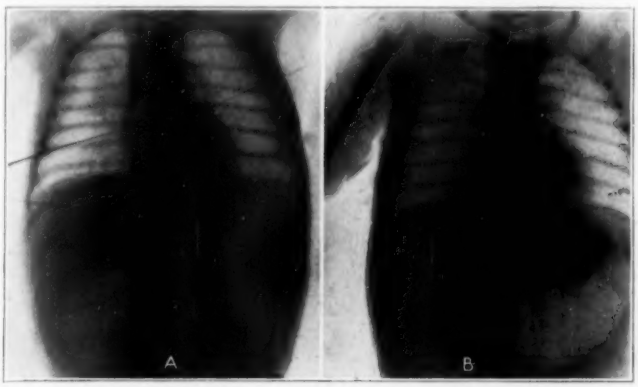


Fig. 3 (case 3).—*A*, spindle-shaped shadow in esophagus slowly emptying into stomach. The picture was taken from three to four minutes after feeding. (The esophagus began to empty from one to two minutes after eating). *B*, taken shortly after *A*, after additional feeding had been given. The shadow is more bottle-shaped.

Examination.—On the fourth day, there was retention of barium in the esophagus for about one hour, with a slight distention of the lower portion. On the sixth day, x-ray examination showed partial retention in the lower part of the esophagus for one and a half hours. The passage of meal through the pylorus was also delayed somewhat. On the thirteenth day, there was no retention in the esophagus after ten minutes. Barium passed through the pylorus in half an hour, with a normal rapid emptying.

Case 3.—*History.*—E., a girl, was delivered easily by forceps, the weight at birth being 4 pounds 6½ ounces (1.9 Kg.). Immediately after the first and subsequent feedings, she vomited everything forcibly through the mouth and nose. The loss in weight was rapid. A feeding of only 5 cc. was vomited at once. Tube feedings, begun at the age of 2½ days, were first retained, but later were also vomited. The child's general condition became extremely bad, with cyanosis, nystagmus and convulsions; the infant died on the fourth day, apparently as a result of cerebral hemorrhage.

Examination.—X-ray examination showed a definite retention of meal in the lower part of the esophagus, with slow passage of food into the stomach. The lower portion of the esophagus showed moderate dilatation.

SYMPTOMS

In older children and adults, the early symptoms are difficulty in swallowing, accompanied by pain or discomfort in the epigastrium and under the lower part of the sternum. This is soon followed by forcible vomiting, occurring during or soon after meals, and characteristically containing food unmixed with gastric juice.

In the conditions observed by me in the new-born infant, vomiting, from the first feeding, which occurs forcibly during or very soon after nursing, is the primary and outstanding symptom. Rapid loss of weight and dehydration fever are the usual result.

Dilatation of the esophagus results in unsuccessfully treated patients, with stagnation and fermentation of the food, if vomiting does not empty the esophagus. Choking, due to the overflow of food from the esophagus into the larynx, is frequent.

DIFFERENTIAL DIAGNOSIS

Numerous conditions responsible for emesis in the new-born infant must be differentiated from cardiospasm, but with the aid of the x-ray picture and fluoroscopy these can usually be readily eliminated. With the addition of a teaspoon of barium to 1 or 2 ounces of breast milk, or a mixture of cow's milk, the feeding can readily be seen to pass through the normal cardiac orifice practically without delay.

In cardiospasm, there is an obvious delay, with definite stasis of part of the meal in the lower portion of the esophagus for varying periods. In the incomplete type, the esophagus fills in an irregular spindle-shaped form, and for a few minutes nothing passes; then a narrow ribbon-shaped stream, also in the form of a spindle, dribbles down the esophagus below the obstruction.

The complete spasm shows a uniform dilatation of the esophagus, with visible contractions above the obstruction. Little if any barium passes through the cardia.

Congenital stricture of the esophagus is rare. Morse [26] reported three cases, in children, who had a history of vomiting from birth, especially of solid food; liquids were swallowed easily.

The roentgenogram demonstrates the strictures, which are almost always in the upper or middle portions of the esophagus. La Fetra described a similar case in 1909.

---

26. Morse, J. L.: Congenital Stricture of the Esophagus, Am. J. Dis. Child. **19**:145 (Feb.) 1920.

Esophageal diverticulae may cause vomiting, and may be differentiated by x-ray examination.

The case of an infant, aged 6 months, was described by Adams in 1909. This child vomited from birth. At autopsy, three diverticulae were found.

Congenital atresia of the esophagus causes incessant vomiting from birth, and is fatal within two weeks. These patients show a stenosis of the middle portion of the esophagus, with the lower part usually entirely absent. Such cases have been reported by Brenneman [27] and by Reynolds and Morrison.[28]

Pharyngospasm, as described by Finkelstein, often accompanies pylorospasm. It is manifested by gagging, when an attempt is made to introduce any object past the pharynx. Both conditions may be associated with cardiospasm.

Spasm of the entire musculature of the stomach, causing severe vomiting from the first feeding, was described by Lust.[29]

Pylorospasm and pyloric stenosis were observed by Greer [30] and others as early as the first week of life, although vomiting does not begin before the third day. The presence of gastric juice in the vomitus points to its origin. X-ray examination here is conclusive.

Cerebral vomiting, accompanying cerebral hemorrhage or other increased intracranial pressure, accompanies other symptoms typical of the condition.

Additional causes of vomiting in the new-born infant which must be borne in mind include: (1) swallowed amniotic fluid, (2) toxemia in the mother, (3) sepsis, (4) obstructed hernias, especially of the diaphragmatic type, (5) hemorrhagic disease, (6) overfeeding, (7) mechanical irritations of fauces from overlarge or long nipples and (8) congenital syphilis. Aballi [31] stated that the latter may cause vomiting on the first day of life.

TREATMENT

In adults, cardiospasm can usually be overcome by dilatation sufficient to cause paralysis of the cardiac sphincter. Walton [16] advised digital dilatation through an incision into the stomach. Mutch,[18] Erdman,[32] Plummer [23] and Sippy all advocated dilatation from above

27. Brenneman, J.: Congenital Atresia of the Esophagus, Am. J. Dis. Child. **16**:143 (Sept.) 1918.

28. Reynolds, R. P., and Morrison, W. W.: Congenital Malformations of the Esophagus, Am. J. Dis. Child. **21**:339 (April) 1921.

29. Lust, F.: Arch. f. Kinderh. **76**:203 (Aug.) 1925.

30. Greer, David: Vomiting in Infants, J. A. M. A. **87**:936 (Sept. 18) 1926.

31. Aballi, A., discussion of Greer (footnote 30).

32. Erdman, J. F.: Cardiospasm, Surg. Gynec. Obst. **14**:286, 1912.

by means of the stomach tube, bougies, hydrostatic bags or mechanical dilators. One or two treatments usually effect a cure. The administration of atropine and benzyl benzoate is occasionally effective if these substances are given in large doses. The diet advised is of bland soft food, given often, in small amounts. Proper hygiene and measures to overcome the nervous elements generally present are important.

In infancy, a few feedings through a catheter serve to relax the spasm and in the mild cases may be permanently successful. Apparently, early diagnosis is advisable, as the treatment is more easily effected during the first few weeks of life.

SUMMARY

1. Cardiospasm in the new-born infant has hitherto escaped early diagnosis.

2. Three cases of cardiospasm occurring in the new-born infant are reported.

3. They present similar symptoms, of which the outstanding is forcible vomiting, beginning with the first feeding, occurring during or soon after nursing and showing no gastric contents in the vomitus.

4. Roentgen examination is simple and definitely positive.

5. The spasm of the cardia is apparently easily overcome by the passage of a catheter through it for a number of feedings.

6. The apparent association with a mild degree of pylorospasm is present in some cases.

CONCLUSION

The condition of cardiospasm in the new-born infant probably exists more commonly than has previously been apparent and should be considered as a possibility in vomiting of early infancy.

ABSTRACT OF DISCUSSION

DR. FRANK C. NEFF, Kansas City, Mo.: In seeking the explanation of early vomiting, one must not overlook important conditions such as cerebral injury, infections of various kinds, including pyelitis, toxemia of the mother and child, organic abnormalities of the esophagus and intestines. As a basis for my discussion, I wish to mention the history of an infant which might fit into the diagnosis of a so-called cardiospasm. A new-born infant, weighing 7 pounds (3.2 Kg.), normal as to birth, mother and development, without any fever, began to vomit by regurgitating the water given him on the first day. Breast milk or formula was regurgitated at two or three of the daily feedings. He was never overfed, but the milk came up unchanged a few minutes after being given. At no time did he take more than 75 cc. per four-hour feeding. Obstruction of the esophagus was ruled out by the ready passage of a catheter into the stomach. The stools were normal, but the child did not regain his birth weight during the new-born period. On the second day, he was given some barium and as it was nursed from the bottle each swallow was observed. The barium collected in the lower half of the esophagus,

for as long as three minutes; if the child were in the recumbent posture, the mediums would move back and forth apparently with each diaphragmatic effort. Eventually, the mediums trickled into the stomach. When the child was held upright, there was little, if any, delay in the mediums entering the stomach. The pylorus emptied promptly. The subsequent history of this child is that he stopped vomiting when he left the hospital, and he gained normally in weight. There was no apparent dilatation of the esophagus, nor was any spastic state demonstrable. There was little difference between this child and a normal new-born infant. From the ease with which air and milk pass the normal esophageal-cardia junction in regurgitation, it is not hard to understand that the new-born infant may lose considerable food if there is any congenital insufficiency of the muscular or nervous mechanism in the stomach and esophagus. I believe my case was of that type.

It is hard to figure out how spasm can localize itself at the cardia, as it has been shown by anatomists that no sphincter muscle is present at the junction of the esophagus and cardia. The compression of the cardia by the diaphragm does produce a pinchlike valve action, and one would not be surprised if the neuropathic child exhibited spasticity of the diaphragm as well as of the entire digestive tube. Infants do seem to have a lack of coordination between the muscles of the esophagus and the stomach. It would seem that for the diagnosis of esophagocardiospasm there should be in addition to the vomiting either a retention in the esophagus or a resistance to the passage of a sound or bougie. From the present knowledge of the anatomy, one would feel that the term cardiospasm should be changed to some other more anatomically appropriate term.

Dr. Ralph M. Tyson, Philadelphia: Many new-born infants show a hyperactivity of their gastro-intestinal tract. This is readily seen by the observation of the abdomen during which peristaltic waves of the stomach and the intestines are frequently seen. The question whether projectile vomiting comes with the obstruction of the lower end of the esophagus alone or is part of this hyperactivity of the gastro-intestinal tract is raised. As Dr. Neff has just stated, it has been shown anatomically that there is no sphincter muscle at the cardiac end of the stomach, and that the obstructions at the lower end of the esophagus are due to the pinchcock action of the diaphragm on the esophagus. He suggested that another name be given to the symptom complex other than cardiospasm. Two years ago I reported a case of similar nature to which I gave the name phrenospasm.

Dr. Birnberg: For a long time I made roentgenograms of all cases of vomiting in children, infants or new-born infants. I was interested in observing if there was a similar spasm of the cardiac orifice in children who had vomiting of a different type, with the pressure of bile, or the type who vomited food containing gastric juice. In those cases I found no evidence of obstruction. Apparently, in the normal child, there was no obstruction in the cardiac region, the food passing through in one stream. As to the expression "cardiospasm," I used that expression for a spasm in the region of the cardiac orifice, not considering its etiologic origin at all, but using an expression commonly used by others for such a condition.

INTRACRANIAL HEMORRHAGE IN THE NEW-BORN INFANT AS DEMONSTRATED BY THE ROENTGEN RAYS*

M. HINES ROBERTS, M.D.
ATLANTA, GA.

Owing to the extreme fragility of the brain tissue in new-born infants, postmortem examination of an intracranial pathologic process often gives difficulty in determining its extent. Usually the point of rupture of the vessels may be found, but the amount of injury produced by the extravasated blood is frequently impossible to determine.

In an effort to obviate this difficulty, Schoenholz [1] suggested that information concerning the pathologic changes of intracranial hemorrhage might possibly be obtained by the injection of some material impermeable to the roentgen rays into the cerebral vessels. He attempted to show this by the use of red lead solutions. He showed in a rather striking manner the normal intracranial circulation, and in three infants dying of intracranial hemorrhage he showed the extent of the pathologic changes by means of the shadow cast by the metal after its extravasation from the vessels.

Appreciating the difficulty of postmortem examinations and further realizing the value of this method in case autopsy was refused, it was determined to attempt some similar studies with certain other metals which seemed likely to lend themselves more readily to such a procedure. Metallic mercury and certain of the iodine-oil suspensions were tried, with the results shown in the accompanying illustrations.

Figure 1 shows the complete arterial circulation of a stillborn infant weighing 2,300 Gm., obtained by injecting 30 cc. of metallic mercury into the right common carotid artery. The cerebral circulation was so sharply defined in this study that it seemed certain that any break which might occur in the vessel wall would be clearly and definitely demonstrated by this method.

The following observations were made on stillborn infants shown by lumbar puncture to have suffered from intracranial hemorrhage, or on infants dying of this condition during the first few days of life, as diagnosed clinically and proved by spinal puncture.

---

* Submitted for publication, July 1, 1929.

* From the Departments of Pediatrics and Obstetrics, School of Medicine, Emory University.

1. Schoenholz, L.: Diagnosis of Intracranial Hemorrhage in Stillborn without Necropsy, Klin. Wchnschr. **5**:1775 (Sept. 17) 1926.

Figure 2 is a roentgenogram of a new-born infant, having the usual clinical evidences of intracranial hemorrhage following prolonged labor and forceps delivery. The spinal fluid was definitely bloody, and the pressure was considerably increased. Death occurred on the third day. Thirty cubic centimeters of metallic mercury was injected into

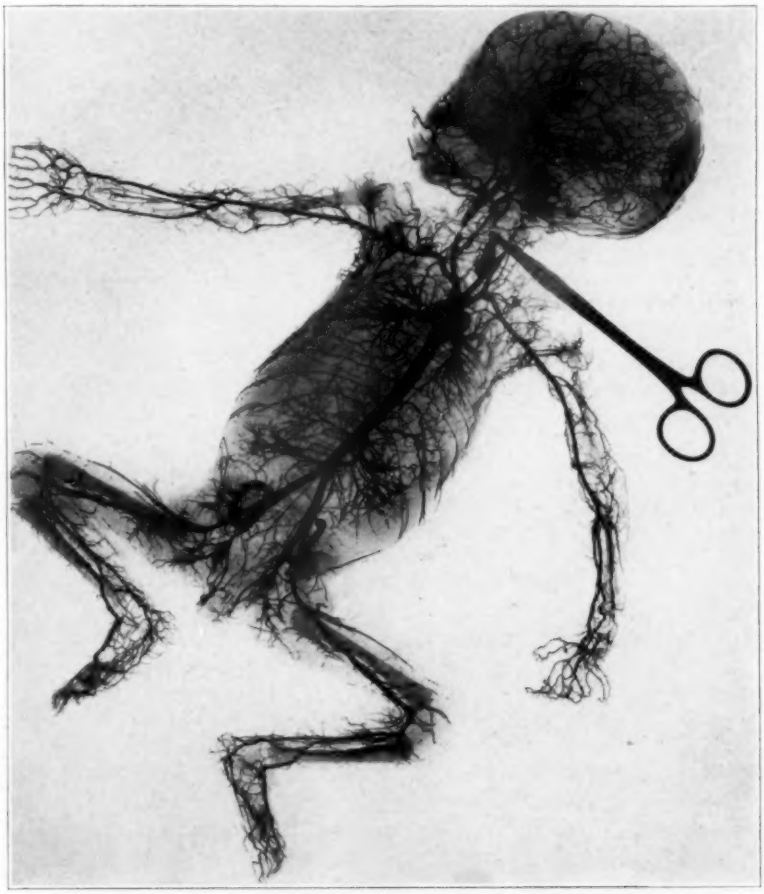


Fig. 1.—Complete circulation of a stillborn infant.

the right carotid artery. The illustration shows large blobs of mercury apparently in the ventricle and over the hemisphere. Autopsy showed hemorrhage into the right ventricle and a large clot over the cerebral hemisphere posteriorly and to the right.

Figure 3 shows the condition occurring in a premature stillborn infant. Lumbar puncture immediately after delivery gave quite a bloody fluid. Thirty cubic centimeters of metallic mercury was injected

Fig. 2.—Roentgenogram of new-born infant, showing large blobs of mercury apparently in the ventricle and over the hemisphere.

Fig. 3.—Roentgenogram of a premature stillborn infant, showing large blobs of mercury apparently in each ventricle and near the base.

Fig. 4.—Roentgenogram of a stillborn infant, showing a large shadow apparently in the ventricle.

Fig. 5.—Roentgenogram of an infant, showing hemorrhage at the base.

into the right carotid. Figure 3 shows large blobs of mercury, apparently in each ventricle and near the base. Autopsy was not obtained.

Figure 4 is a roentgenogram of a stillborn infant whose spinal fluid, taken immediately after a long hard labor, was very bloody and under greatly increased pressure. Thirty cubic centimeters of metallic mercury was injected into the right carotid. A large shadow apparently in the ventricle may be seen. Autopsy revealed a massive clot in the left ventricle and a smaller one in the right.

The infant shown in figure 5 never breathed well, was cyanotic and died a few hours after birth. Lumbar puncture gave a very bloody fluid with markedly increased pressure. Thirty cubic centimeters of iodized oil was injected into the aorta, with the resulting picture

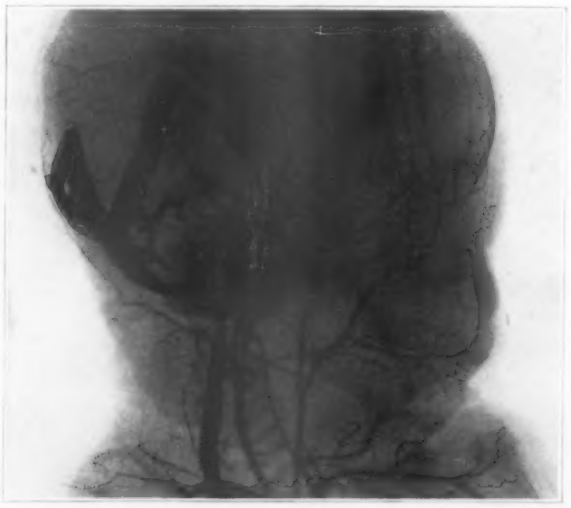


Fig. 6.—Study of a premature stillborn infant. Small hemorrhages are present through the hemisphere and at the base.

apparently showing hemorrhage at the base. Autopsy showed a tentorial tear with extensive subtentorial hemorrhage.

Figure 6 is the study of a premature stillborn infant whose spinal fluid showed evidence of extensive intracranial hemorrhage. Thirty cubic centimeters of iodized oil was injected into the ascending aorta. There are apparently small hemorrhagic areas through the hemisphere and at the base. Autopsy was not obtained.

The metallic mercury has the great advantage of definite outline due to the marked contrast. However, there seems to be one serious disadvantage; owing to the weight of the substance, there is the tendency always to seek the lowest level. Any manipulation after injection causes some shifting of the free mercury and consequently may give

an erroneous impression of the location and even the extent of the hemorrhage. Iodized oil, on the other hand, does not cast the sharply outlined shadow of metallic mercury, but probably shows more accurately the extent and true location of the hemorrhage.

These observations seem to show that some valuable information may be obtained by means of the injection of certain metals into the arterial circulation of new-born infants suffering with intracranial hemorrhage. It is of peculiar value in cases in which an autopsy is not permitted.

ANOMALIES OF THE URINARY TRACT IN INFANTS *

ALGERNON S. HURT, JR., M.D.
Fellow in Pediatrics, the Mayo Foundation
ROCHESTER, MINN.

Urologists and pediatricians are realizing the importance of determining the presence of anomalies of the urinary tract in infants and in children, and various investigators, among whom are Abt,[1] Helmholz,[2] Hunner,[3] Hyman,[4] Kretschmer,[5] Lowsley and Butterfield,[6] McKay,[7] Mertz [8] and Stevens,[9] have emphasized the necessity of making complete urologic examinations of children who have urinary trouble which is resistant to intensive treatment over a reasonable period of time. The application of modern urologic methods has made it possible to diagnose an increasing number of urinary anomalies in infants and in children during life, instead of waiting for necropsy to reveal them. The actual frequency of urinary anomalies cannot be determined by a consideration of clinical diagnoses, since only those patients whose anomalies are causing trouble consult a physician.

---

* Submitted for publication, July 27, 1929.

* From the Section on Pediatrics, the Mayo Clinic.

* Read before the Northwestern Pediatric Society, Rochester, Minn., July 20, 1929.

1. Abt, I. A.: Pyuria in Malformations of the Urinary Tract: A Review of the Literature, Am. J. Dis. Child. **32**:507 (Oct.) 1926.

2. Helmholz, H. F.: Congenital Abnormalities of the Urinary Tract in Childhood, J. A. M. A. **89**:1932 (Dec. 3) 1927.

3. Hunner, G. L.: Ureteral Stricture and Chronic Pyelitis in Children, Am. J. Dis. Child. **34**:603 (Oct.) 1927.

4. Hyman, A.: Surgical Diseases of the Urinary Tract in Children, Am. J. Dis. Child. **15**:116 (Feb.) 1918; Clinical Aspects of Surgical Diseases of the Urinary Tract in Children, J. Urol. **16**:259, 1926.

5. Kretschmer, H. L.: Urologic Problems in Infancy and Childhood, J. Urol. **18**:433, 1927; Urologic Problems in Infancy and Childhood, Am. J. Dis. Child. **33**:952 (June) 1927.

6. Lowsley, O. S., and Butterfield, P. M.: Urologic Conditions Among Children, J. Urol. **16**:415, 1926.

7. McKay, H. W.: The Application of Modern Urologic Diagnostic Methods in Pediatrics: Case Reports, South. M. J. **19**:460, 1926.

8. Mertz, H. O.: Congenital Changes in the Urinary Organs as They Influence Pyuria of Infancy and Childhood, J. Urol. **19**:371, 1928.

9. Stevens, W. E.: Diagnosis and Treatment in Pathologic Conditions of the Urinary Tract in Children: Modern Methods, J. A. M. A. **77**:1081 (Oct. 1) 1921.

On the other hand, data from necropsy in a consecutive series of patients dying from various causes should give a fairly accurate estimate of the number of anomalies of the urinary tract likely to be met with in clinical practice, whether or not they are diagnosed during life. The percentages of anomalies found by different workers at necropsy have varied somewhat. This variation probably depends, to some extent, on the age group included in the series under consideration, and also on whether special attention was paid to the urinary tract during examinations. These figures are also influenced by the types of cases in the series. In a hospital with a good urologic staff there is likely to be recorded a larger proportion of urinary disorders than there is in one without an expert urologist. Bugbee and Wollstein,[10] in 1924, reported the occurrence of anomalies of the urinary tract as 2.3 per cent in a series of 4,903 necropsies done as a routine on children. Of the 117 patients with anomalies in their series, 47 were less than 3 months of age; 69, less than 6 months, and 101, less than 1 year. In 1928, Bigler [11] reported 20 cases, (13 per cent) of anomalies of the urinary tract, in a consecutive series of 153 necropsies on children, in which the pathologist paid special attention to the urinary tract. The ages of those with anomalies ranged from 13 days to 8 years. Campbell and Lyttle,[12] in a recent publication, reported gross evidence of ureteral obstruction in 2 per cent of 2,420 pediatric cases in which necropsy was done. The ages of those with obstruction ranged from the seventh month of fetal life to the sixth year of life; in more than 80 per cent the patients were less than 2 years of age.

The present report is of a study made at necropsy of 101 infants, of whom 51 were males and 50 females. Sixty-three were stillborn; the remainder lived for periods of from a few minutes to twelve days. The necropsies were consecutive and were performed in the pathologic service at the University of Minnesota Medical School from July 1, 1928, to Jan. 7, 1929. The material was obtained from 15 different hospitals in Minneapolis and St. Paul. Anomalies of the urinary tract were found in 5 cases (5 per cent). In 2 of these there were bilateral polycystic kidneys; in 2, unilateral ureteral stricture with hydronephrosis, and in 1 case, absence of one kidney, and the other kidney was small and had been nonfunctioning. Microscopic examination of the kidneys did not reveal evidence of infection.

---

10. Bugbee, H. G., and Wollstein, Martha: Surgical Pathology of the Urinary Tract in Infants, J. A. M. A. **83**:1887 (Dec. 13) 1924.

11. Bigler, J. A.: Anomalies of the Urinary Tract in Children, unpublished data.

12. Campbell, M. F., and Lyttle, J. D.: Ureteral Obstruction in Infancy; a Study of Seventy-Four Cases, J. A. M. A. **92**:544 (Feb. 16) 1929.

REPORT OF NECROPSIES

CASE 1.—The subject was a premature, female infant, aged 45 minutes. The length from crown to rump was 27.5 cm. and from crown to heel, 39 cm. The weight was 1,590 Gm. The cause of death was prematurity. There were bilateral polycystic kidneys; the right kidney weighed 15 Gm. and the left kidney, 12 Gm. Both were completely cystic, and the pelves were small. The remainder of the urinary tract was normal.

CASE 2.—The subject was a premature male infant, aged 6 hours. The length from crown to rump was 27 cm. and from crown to heel, 38 cm. The weight was 1,250 Gm. The cause of death was intracranial hemorrhage and prematurity. There was a partial stricture of the left ureter 1 cm. above its juncture with the wall of the bladder. Mild degrees of hydro-ureter and hydronephrosis also were present. The left ureter was 5 mm. in diameter, and the right, only 2 mm. There was, also, definite narrowing of the left ureter about 2 cm. below the kidney. Each kidney weighed 6 Gm. The remainder of the urinary tract was normal.

CASE 3.—The subject was a premature male infant, aged 3 hours. The length from crown to rump was 30 cm. and from crown to heel, 42 cm. The weight was 1,515 Gm. Although this infant was considerably premature, the anomaly of the urinary tract probably contributed, to a large degree, in causing death. Both kidneys were polycystic, and there was complete stricture of each ureter just below the kidneys. The ureters were patent from the strictures to the bladder, but were delicate and thin-walled. The right kidney weighed 4.2 Gm. and the left, 4.7 Gm. The remainder of the urinary tract was normal.

CASE 4.—The subject was a stillborn, premature male infant. The length from crown to rump was 30.5 cm. and from crown to heel, 42 cm. The weight was 2,000 Gm. Aside from prematurity, the cause of death was not determined, although the urinary anomaly in this case also was such as to be incompatible with the continuance of life for an appreciable length of time. The right kidney and ureter were absent. The left kidney weighed only 2 Gm. and appeared to be a fibrous mass; a pelvis was not demonstrable. The left ureter also was small and delicate; it was obliterated just below the kidney, but was patent from the point of obliteration to the bladder. The remainder of the urinary tract was normal.

CASE 5.—The subject was a premature female infant, aged 10 days. The length from crown to rump was 31 cm. and from crown to heel, 45 cm. The weight was 1,650 Gm. The cause of death was a defect in the interventricular septum. There was almost complete stricture of the left ureter just below the renal pelvis, with marked left hydronephrosis. The pelvis was about five times its normal size, and the cortex was considerably thinned. The left kidney weighed 16.3 Gm., and the right, 10.2 Gm. The remainder of the urinary tract was normal.

COMMENT

Cases 3 and 4 may be eliminated from consideration of anomalies that might be met with later in life, as the urinary apparatus was completely nonfunctioning. In the final analysis, 3 per cent of anomalies in this series is significant. However, due to the variation in types of anomalies, and the comparative rarity of the types found in cases 3 and

4, it is thought that 5 per cent is a fair estimate of the frequency with which anomalies of the urinary tract might be expected in early infancy.

The restriction of this study to infants less than 2 weeks of age eliminates the possible effect on the results of including cases of widely different ages, and the percentage of anomalies represents those found in the urinary tract at birth. It is, therefore, hard to compare the data with those of larger series containing a wider range of ages. However, in all series the percentages are startlingly high, considering the relatively small number of cases that are being found clinically. The figures would seem to indicate either that clinicians are overlooking a good many of these cases, or that many of the anomalies do not cause enough trouble to require treatment.

SUMMARY

In a series of 101 necropsies on infants less than 2 weeks of age, 5 cases (5 per cent) were found in which there were anomalies of the urinary tract.

Since these anomalies occurred in such a low age group, this percentage may be taken as at least an indication of the incidence of anomalies of the urinary tract at birth.

Although the series reported is comparatively small, the data corroborate the evidence that anomalies of the urinary tract are by no means infrequent in infants and children.

THE KAHN PRECIPITATION TEST IN INFANCY AND IN EARLY CHILDHOOD

A COMPARATIVE CLINICAL STUDY WITH THE NOGUCHI-WASSERMANN REACTION *

JOHN CAFFEY, M.D.
AND
KATHERINE V. KREIDEL, A.B.
NEW YORK

The value of the Kahn test in the serodiagnosis of syphilis has been abundantly demonstrated since its introduction in 1922.[1] Kahn,[2] in 1927, compiled reports on 300,000 serums. Comparatively few observations have been made, however, in infantile and juvenile syphilis. The purpose of this report is to present observations on the sensitivity and the specificity of the Kahn test in infancy and in childhood, a comparatively neglected field.

We are in accord with Stokes [3] that complete detailed clinical information concerning patients from whom serums are taken is of first importance in comparative studies of the Kahn and Wassermann tests. There has been intimate clinical contact with all patients herein reported. Our aim has been to determine the results of both Kahn and Wassermann tests in the presence and in the absence of clinical syphilis in the patient, rather than to compare the two tests with each other.

To date, the most comprehensive report of the Kahn test in congenital syphilis is that of McLean,[4] who examined serums from patients at the Hospital for Sick Children, London. He found the Kahn test considerably more sensitive than the Wassermann test in the case of children undergoing treatment for syphilis. He reported no false positive Kahn reaction and no instances in which the Kahn test was negative and the Wassermann test definitely positive in the presence of syphilitic infection. Keim and Kahn,[5] at the University of Michigan, concluded

* Submitted for publication, June 26, 1929.

* From the Babies' Hospital and the Department of the Diseases of Children, College of Physicians and Surgeons, School of Medicine, Columbia University.

* Read before the New York Academy of Medicine, May 9, 1929.

1. Kahn, R. L.: A Simple Quantitative Precipitation Reaction for Syphilis, Preliminary Communication, Arch. Dermat. & Syph. **5**:570 (May) 1922.

2. Kahn, R. L.; Kenderick, P. L., and Landau, J. L.: Report on 300,000 Kahn Tests, J. A. M. A. **89**:84 (July 9) 1927.

3. Stokes, J. H., and Chambers, S. O.: Am. J. M. Sc. **174**:864, 1927.

4. McLean, D.: Observations on the Relative Value of the Wassermann and Kahn Reactions in Cases of Congenital Syphilis, J. Path. & Bact. **30**:182, 1927.

5. Keim, H. L., and Kahn, R. L.: Studies on the Kahn Reaction for Syphilis: I. Diagnostic Test, Arch. Dermat. & Syph. **10**:722 (Dec.) 1924.

from an analysis of thirty-nine cases of congenital syphilis that "the Kahn test is slightly more sensitive than either Wassermann test (12 hour ice box fixation or 1 hour ice box fixation)." Others [6] reporting results in small groups of patients with congenital syphilis gave identical results with the two tests. Keim and Kahn,[7] in a study of the specificity of the Kahn test, found that many of the febrile diseases frequently encountered in infancy and childhood (pneumonia, measles, chickenpox, etc.) do not cause false positive Kahn reactions. Fox and Sanderson's [8] results in fifteen cases of congenital syphilis were slightly favorable to the Wassermann test. Greenbaum [9] cited fifty cases in which the Kolmer-Wassermann test was positive and the Kahn test negative, and also fifty cases in which the Kolmer-Wassermann test was negative and the Kahn test positive. There were nine cases of congenital syphilis in the first group and eight cases of congenital syphilis in the latter group. McIntyre and Gilman,[10] in a study at Stokes' clinic in Philadelphia, reported an absolute disagreement of 28.8 per cent in 111 tests in twenty-two patients with prenatal syphilis; 24.3 per cent were favorable to the Kolmer-Wassermann test and 4.5 per cent were favorable to Kahn's test. However, only Kahn readings of 244 (total of 10) and above were considered positive by them, which may possibly explain part of the wide discrepancies between results of the two tests and the superiority of the Wassermann test in the hands of these workers. Kahn advised that a reading of a total of 6 and above should be considered as a strong serologic indication of the presence of syphilis.[11] We have followed this interpretation of readings (table 1).

The Wassermann test used in this study is the standard Noguchi modification.[12] The antigen and hemolytic amboceptor were generously

---

6. Faupel, M. H.: A Comparison of Kahn with Wassermann Test, Bull. Johns Hopkins Hosp. **37**:170, 1925. Kelly, R. L.: Clinical Study of the Kahn Precipitation Test and Kolmer Complement Fixation Test, Arch. Dermat. & Syph. **12**:720 (Nov.) 1925.

7. Keim, H. L., and Kahn, R. L.: Clinical Studies on the Kahn Reaction for Syphilis: Specificity of the Test, J. Lab. & Clin. Med. **10**:1013, 1925.

8. Fox, J. C., Jr., and Sanderson, E. S.: Observations on the Kahn Precipitation Test for Syphilis, Am. J. Syph. **12**:687, 1923.

9. Greenbaum, S. S.: The Error of Basing Serum Diagnosis on the Kahn Reaction Alone, J. A. M. A. **86**:1273 (April 24) 1926.

10. McIntyre, M. D., and Gilman, R. L.: A Clinical Comparison of the Kolmer Modification of the Wassermann Test and the Kahn Precipitation Test in Syphilitic and Non-Syphilitic Cases, Am. J. M. Sc. **186**:843, 1928.

11. Kahn, R. L.: The Kahn Test a Practical Guide, Baltimore, Williams & Wilkins Company, 1928, p. 130.

12. Noguchi, H.: Laboratory Diagnosis of Syphilis, New York, Paul B. Hoeber, Inc., 1923.

furnished us by the late Dr. Noguchi. We wish to emphasize the fact that this series of cases does not present comparisons of the Kahn test with other Wassermann technics (special antigens, icebox fixations, Kolmer modification, etc.), but only with the standard Noguchi-Wassermann test. Serums were examined on the day of withdrawal from the patient, and usually within three hours after venipuncture. Four plus and three plus reactions were considered positive; two plus and one plus were considered doubtful.

The standard Kahn technic [13] was carried out in every detail. The antigen used throughout was prepared in Kahn's laboratory. Both tests have been done throughout this study by one person (K. K.). The same is true of all readings which have been repeated on successive days. The specimens of blood were withdrawn from either the cubital or the jugular veins.

TABLE 1.—*Interpretation of Results of Kahn Test*

Test Number	Reaction in Tube 1	Reaction in Tube 2	Reaction in Tube 3	Final Result	
1	4	4	4	4 plus	
2	3	4	4	4 plus	
3	2	4	4	3 plus	
4	1	4	4	3 plus	
5	0	4	4	3 plus	
6	1	3	4	3 plus	Positive
7	0	3	4	2 plus	
8	0	3	3	2 plus	
9	1	2	3	2 plus	
10	0	2	4	2 plus	
11	0	1	4	2 plus	
12	All precipitation below this			Doubtful	
13	0	0	0	Negative	

During the thirty-three months (from March, 1926, to January, 1929) at the Babies' Hospital, 1,729 serums of infants, young children and mothers have been examined with parallel Wassermann and Kahn tests. These serologic examinations have been in no sense a routine procedure, but have been made only on the serums of patients in whom there was some evidence, historical or physical or both, which made serologic examination for syphilis desirable.

For the purpose of clarity, we believe this material can best be grouped into three main divisions: 1,185 infants and young children under investigation for syphilis; 43 patients under treatment for syphilis; 315 mothers of patients, in the aforementioned groups.

WASSERMANN AND KAHN TESTS IN EXAMINATION OF YOUNG CHILDREN FOR SYPHILIS

These 1,185 patients were studied clinically by careful taking of the family history, by physical examination and, when indicated, by exam-

13. Kahn (footnote 11, p. 111).

ination of the spinal fluid, roentgenologic examination for osteal lesions, dark-field examination for spirochetes in suitable cases, and serologic examinations of available members of the family. In 104 cases, these investigations warranted a clinical diagnosis of syphilis, and in 1,081, there was not sufficient clinical evidence to support a diagnosis of syphilis.

The serologic results in the clinically syphilitic group are shown in table 2. Group A includes seventy-six patients younger than 1 year, forty-six of whom are 3 months of age or younger. Syphilis in the first months of life furnishes one of the most reliable clinical checks for serologic reaction because of the certainty with which the clinical diagnosis can be made. This is true because of the high incidence of bone lesions demonstrable by x-ray, a highly diagnostic skin eruption and the usual availability of mothers for confirmatory investigation of maternal

TABLE 2.—*Results of Serologic Tests in One Hundred and Four Cases of Clinical Congenital Syphilis*

Group A, 76 Patients Younger than 1 Year: 46 under 3 Months Old; 20 from 3 to 6 Months Old; 10 from 6 to 12 Months Old						Group B, 28 Patients Older than 1 Year: 17 from 1 to 3 Years Old; 11 Older than 3 Years					
Results in Agreement			Results in Disagreement			Results in Agreement			Results in Disagreement		
Wassermann	Kahn	Serums	Wassermann	Kahn	Serums	Wassermann	Kahn	Serums	Wassermann	Kahn	Serums
+	+	67	+	0	1	+	+	22	+	0	2
0	0	1	+	Doubtful	1				+	Doubtful	2
Doubtful	Doubtful	0	0	+	1				0	+	2
Total.....		68	Anticomplementary	+	5				Total.....		6
			Total.....		8						

syphilis. In this group, Wassermann test and Kahn test were in complete agreement in sixty-eight cases (positive, 67; negative, 1) and were in disagreement in eight cases.

Details of the single case in which both tests were negative and of the eight cases in which there were discrepancies (table 2) are of value.

Both Kahn Test and Wassermann Test Negative in Frank Clinical Syphilis.—The serum of S. J., aged 9 weeks, gave a Wassermann reaction of 0 and a Kahn reaction of 000. Investigation of the family revealed that the mother had been previously diagnosed syphilitic. Her serology checked by us gave: Wassermann, 4; Kahn, 444. The infant presented a typical cutaneous syphilis of early infancy, and roentgenograms of the long bones showed a specific osteochondritis. One injection of sulpharsphenamine, 0.05 Gm., was given intramuscularly. Three weeks later, when the child was 12 weeks old, a reexamination showed a reversal to a double positive: Wassermann, 4; Kahn, 444.

The occasional presence of active syphilis and a negative Wassermann reaction in the first weeks of life, subsequently becoming positive

under treatment, has been commented on by Fordyce and Rosen.[14] Our single such case suggests that the Kahn test is also negative under similar conditions and follows the Wassermann both in early negativity and reversal after treatment.

Discrepancies Between Results of the Parallel Tests in Syphilitic Infants Younger Than 1 Year.—A Wassermann reaction positive and a Kahn reaction negative, constituting an absolute discrepancy, was found in one case. The serum of G. G., aged 5 months, gave a Wassermann reaction of 4 and a Kahn reaction of 000. Maternal syphilis had been diagnosed ante partum, and antisyphilitic therapy had been administered for four months of the pregnancy. The mother's serology five months post partum was Wassermann, 1; Kahn, 444 (the inversion of the maternal and the infantile serology should be noted). Clinically, this patient presented rhagades, cutaneous syphilids, enlarged epitrochlear glands and marasmus (weight 6 pounds, 3 ounces [2.8 Kg.] at the fifth month). Three intramuscular sulpharsphenamine treatments were given, and at 6½ months the serology was Wassermann, 4; Kahn, 003."

A Wassermann reaction positive and a Kahn reaction doubtful, constituting a relative discrepancy, was observed in one case. The serum of E. K., aged 1 month, gave a Wassermann reaction of 4 and a Kahn reaction of 011. The father was known to be syphilitic. The maternal serology one month post partum was Wassermann, 0; Kahn, 000. No syphilitic manifestations could be detected by physical examination. Necropsy, however, showed undoubted syphilitic osteochondritis.

A Wassermann reaction negative and a Kahn reaction positive, constituting an absolute discrepancy, was observed in one case. The serum of U. H., aged 2 months, gave a Wassermann reaction of 0 and a Kahn reaction of 144. The family history was negative for syphilis. Also, the mother was negative serologically: Wassermann, 0; Kahn, 000. The infant had marked snuffles and a definite desquamation of the palms and soles. Roentgenograms of the long bones showed osteal syphilis. No recheck of this infant's serology was possible.

The Wassermann reaction was anticomplementary and the Kahn reaction positive in five cases. The oldest of these patients was 9 months of age, one was 4 months and three were 2 months of age. They all had definite clinical syphilis. In the four younger patients, the Kahn reaction was 444; in the oldest (9 months) the Kahn reaction was 122. The absence of irregularities in the Kahn test in serums which fix complement nonspecifically (anticomplementary reactions) is an important practical advantage of the precipitation test which has been consistently observed by many workers in serology. Its importance in early

14. Fordyce, J. A., and Rosen, I.: Results in the Treatment of Congenital Syphilis, Arch. Dermat. & Syph. **9**:355 (March) 1924.

life will be appreciated in the fact that anticomplementary serums averaged one in every fifteen syphilitic serums examined from our patients younger than 1 year. We have not recorded the anticomplementary reactions in the serology of patients clinically free from syphilis.

Discrepancies Between Results of the Parallel Tests in Clinically Syphilitic Children Older Than 1 Year.—The results in tests on twenty-eight clinically syphilitic patients older than 1 year are also shown in table 2, group B. Nineteen of these were younger than 3 years. The clinical diagnosis in this group is not so certain as in earlier life because of the usual subsidence of the active infantile phenomena, such as osteochondritis, the early eruption and snuffles. In this group, the clinical diagnosis has been based in several instances on such lesions as interstitial keratitis, Hutchinsonian teeth and tertiary nodulo-ulcerative syphilids. We have also considered cases in which there were no physical manifestations of the disease but in which there were a familial history and a parental serology indicative of the presence of familial syphilis, clinically positive if the reaction in either the Kahn or the Wassermann test or in both tests was definitely positive. Also patients in this group have been considered syphilitic who showed repeated positive reactions in both tests, even though there was no clinical or familial evidence to support the diagnosis. This has, of course, arbitrarily eliminated the possibility of repeated simultaneous false positive reactions in both tests. Of the twenty-eight syphilitic patients older than 1 year, twenty-two had positive readings in both tests, and there were 6 serums in which the tests were at variance. There were no patients clinically syphilitic in whose serums both tests were negative.

The details of the six instances of discrepancies (table 2) are as follows:

The Wassermann reaction was positive and the Kahn reaction negative, constituting an absolute disagreement in two cases. The serum of D. Y., aged 26 months, gave a Wassermann reaction of 4 and a Kahn reaction of 000. The family history was definitely positive. The father had been treated for syphilis two years before. The mother had had weekly injections for syphilis over a period of six months during this pregnancy. The maternal serology was Wassermann, 0; Kahn, 000. There were no physical signs of syphilis in this patient.

The serum of F. M., aged 1 year, gave a Wassermann reaction of 4 and a Kahn reaction of —0. Three weeks later the following results were obtained: Wassermann, 4; Kahn, —4. The maternal serology was Wassermann, 4; Kahn, 444. This was an undoubted case of syphilis of the meninges with convulsions and hydrocephalus. The spinal fluid showed increased protein, a cell count of 70 and a Wassermann reaction of 4.

The Wassermann reaction was positive and the Kahn reaction doubtful, amounting to a relative disagreement, in two cases. The serum of E. T., aged 24 months, gave a Wassermann reading of 4 and a Kahn reading of 011. The mother gave a history of one stillbirth, and also of premature twins who died on the fifth day. The maternal serology was Wassermann, 4; Kahn, 000. The child was given, weekly, sulpharsphenamine alternating with courses of bismuth bitartrate. After three and a half months of treatment, the serology showed Wassermann, 2; Kahn, 112. After four and a half months, the Wassermann reading was 0; the Kahn, 012. There were no physical signs of syphilis.

One could, perhaps, demand more evidence to establish the diagnosis of syphilis in this patient. The positive Wassermann reaction in the mother's serum and the appearance of a Kahn reaction of 112 after three and a half months were factors that seemed to justify the diagnosis of syphilis in this borderline case.

The serum of C. R., aged 19 months, gave a Wassermann reaction of 4 and a Kahn reaction of 001. An obstetric clinic reported the mother's blood positive during pregnancy, and she was said to have had "three treatments in the arm." The maternal serology was Wassermann, 2; Kahn, 112. There were no physical signs of syphilis in this patient.

The Wassermann reaction was negative and the Kahn reaction positive, an absolute disagreement, in two cases. The serum of J. G., aged 8 years, gave a Wassermann reading of 0 and a Kahn reading of 222. There was definite familial syphilis. The maternal serology was Wassermann, 4; Kahn, 444. A younger brother had frank infantile syphilis, with a Wassermann reaction of 4 and a Kahn reaction of 444. There were no physical signs of syphilis in the patient. The serum of J. S., aged 4 years, gave a Wassermann reading of 0 and a Kahn reading of 444. The family history was definitely syphilitic. The mother had had a primary sore previous to this pregnancy; her serology was Wassermann, 0; Kahn, 244 (the parallelism of the negative Wassermann reactions in child and mother should be noted. A younger brother had a Wassermann reading of 4; a Kahn of 444. There were no physical evidences of the disease in the patient.

Comment on Comparative Sensitivity and Specificity.—The clinical comparison of the two tests as regards their sensitivity in serum from 104 infantile and juvenile clinically syphilitic patients is shown in table 3. One is impressed by the parallelism of the results, ninety being in complete agreement, and the lack of a decided advantage in either. Of fourteen discrepancies, six are favorable to the Wassermann test and eight are favorable to the Kahn test. The cases in absolute disagreement (one test positive and the other negative) are equally divided, three being favorable to the Wassermann test and three being favorable to the

Kahn test. These are of considerable importance, because one test definitely indicates syphilis, while the other is wholly negative. The Wassermann test shows a definite advantage in three cases of relative disagreement, in which it gave clear-cut positive results, while the Kahn results were weakly positive. However, the presence of clearcut Kahn reactions in five serums in which the Wassermann reactions were anticomplementary would seem slightly to outweigh the three cases of relative disagreement shown by the Wassermann test. One should keep in

TABLE 3.—*Comparative Sensitivity of Wassermann and Kahn Tests in One Hundred and Four Cases of Clinical Congenital Syphilis*

Results of Tests	Serums	Percentages
Results in agreement	90	86.55
Results in disagreement	14	13.45
Wassermann, +; Kahn, 0	3 } 6 cases favorable to Wassermann	
Wassermann, +; Kahn, doubtful	3 } test (5.76%)	
Wassermann, 0; Kahn, +	3 } 8 cases favorable to Kahn	
Wassermann, anticomplementary; Kahn, +	5 } test (7.69%)	
Both tests negative in the presence of active infantile syphilis in 1 case		
		Percentages
Sensitivity of Wassermann test		91.34
Sensitivity of Kahn test		93.03

TABLE 4.—*Comparative Specificity of the Wassermann and Kahn Tests in One Thousand and Eighty-One Patients Clinically Free from Syphilis*

A. Both Tests Negative 1,025 (91.8%)	B. One or Both Tests Doubtful 26 (2.4%)			C. One Test Positive; Other, Doubtful or Negative 30 (2.8%)		
	Wassermann	Kahn	Serums	Wassermann	Kahn	Serums
Approximately 50 per cent were infants; acute, as well as chronic, diseases, febrile and afebrile, were represented; practically all the patients were younger than 5 years	Doubtful	0	13	+	0	9
	0	Doubtful	8	+	Doubtful	4
	Both tests doubtful		5	0	+	17
	Examination was repeated in nine cases, three being those in which results in both tests were doubtful; all showed double negatives in the later examinations			Only two patients younger than 6 months; four younger than 1 year. Repeated examinations on seven gave double negatives. Asymptomatic syphilis? False positive serology?		

mind that occasionally in the early weeks of life (one case in this series) both tests may be negative in infants with active syphilis. Both tests proved highly sensitive and sensitive in almost equal degree: Wassermann, 91.34 per cent; Kahn, 93.03 per cent.

The determination of the comparative specificity of each test is shown in table 4. The 1,081 patients included here were investigated clinically and were judged clinically free from syphilis. The parallel serologic tests divide themselves into three groups: A, both tests negative; B, one or both tests doubtful; C, one test positive and the other doubtful or negative.

Group A, both tests negative, represents both tests in negative agreement in cases clinically free from syphilis. One thousand twenty-five, or 94.8 per cent of the patients clinically free from syphilis were entirely negative serologically in both tests. Slightly more than half of these patients were younger than 1 year. They represented practically all of the acute and chronic febrile and afebrile diseases commonly met with in an active hospital and outpatient service in New York City, and also well infants and children. It is apparent that early age and the common infantile and juvenile diseases do not interfere materially with the specificity of either reaction.

Group B (table 4), one or both tests doubtful, represents twenty-six patients (2.4 per cent) whose serums registered weakly positive reactions in one or both tests. These false, weakly positive reactions occurred slightly more frequently with the Wassermann test than with the Kahn test. Repeated examinations of nine of these serums gave double negatives. Three of the five patients whose serums showed doubtful reactions in both tests were rechecked, and the results were negative in both the Wassermann and the Kahn tests at the second examination, also, which indicates that even weak reactions in both tests, if unsupported clinically, should be given little diagnostic significance.

Group C (table 4), one test positive and the other doubtful or negative, is of more importance than the previous group because the one test was strongly positive in the absence of any clinical evidence. The Wassermann test and the Kahn test showed an absolute disagreement in twenty-six cases and a relative disagreement in four cases, a total of thirty, or 2.8 per cent of all the cases clinically free from syphilis. Only two of the patients in these cases were younger than 6 months and four were younger than 1 year. Repeated examinations of seven of these serums gave double negatives, which suggests that the first results may have been false positive reactions. Longer periods of observations, however, are necessary to determine the true significance of the results in this group; that is, whether they represent cases of asymptomatic syphilis with discrepancies in the serology or whether they represent cases free from syphilis, the serologic reactions being falsely positive.

There are no instances of simultaneous positive reactions in both Wassermann and Kahn tests in nonsyphilitic patients.

A summation of the three groups in table 4 gives a Wassermann specificity of 98.8 per cent and a Kahn specificity of 98.1 per cent.

WASSERMANN AND KAHN TESTS IN EXAMINATION OF PATIENTS UNDER TREATMENT FOR SYPHILIS

The serologic results with both tests in serums of patients under treatment are shown in table 5. One hundred fifty-four serums from

forty-three patients were examined. There is complete agreement of the two tests in 123 (79.9 per cent), and in thirty-one (20.1 per cent) a relative or absolute disagreement. In the absence of clinical signs in treated patients, it is perhaps not possible to determine conclusively the presence or absence of active or potential syphilis when the serologic results are in disagreement. We have attributed a superior sensitivity to the test that was the more positive in all treated cases in which the serologic reactions disagree. With such a rating, the thirty-one dis-

TABLE 5.—*Comparative Sensitivity of the Wassermann and Kahn Tests in Forty-Three Patients Under Treatment for Syphilis*

Results in Agreement			Results in Disagreement			
Wassermann	Kahn	Serums	Wassermann	Kahn	Serums	Comparative Sensitivity
+	+	55	+	Doubtful	2	5 cases favorable to Wassermann test
Doubtful	Doubtful	4	+	0	2	
0	0	64	Doubtful	0	1	
Total.....		123	Doubtful	+	9	26 cases favorable to Kahn test, indicating superiority of sensitivity of Kahn test
			0	+	17	
			Total.....		31	

The scarcity of doubtful reactions in both tests should be noted

TABLE 6.—*Two Examples of Exact Serologic Parallelism During Early Complete Reversal During Treatment*

Anna M., 9 Weeks			Ralph D., 3 Months		
	Wassermann	Kahn		Wassermann	Kahn
Before treatment..	4	444	Before treatment..	4	444
Period treated			Period treated		
2½ months.....	4	444	2 weeks.....	4	444
7½ months.....	0	000	5 months.....	0	000
12 months.....	0	000	9 months.....	0	000
16 months.....	0	000	11 months.....	0	000
20 months.....	0	000	14½ months.....	0	000
			20½ months.....	q.n.s.*	000
			26½ months.....	0	000

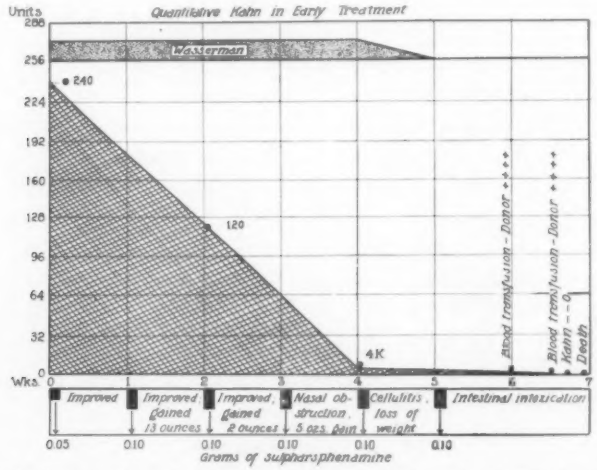
* q.n.s. = quantity not sufficient.

crepancies of this group show five results favorable to the Wassermann test and twenty-six to the Kahn test. The superiority of the Kahn sensitivity in treated cases thus approximates a 5 to 1 ratio in the serums in disagreement. The evidence here suggests, quite markedly, that positive Wassermann reactions in infancy and childhood are more easily reversed by antisyphilitic therapy than are positive Kahn reactions. The paucity of doubtful reactions in both tests in this treated group is noteworthy.

Table 6 shows results in two treated patients who responded with rapid complete serologic reversal in which both tests paralleled one another throughout the reversal period and persisted negative following

reversal. These are two of eleven such patients seen during the last three years. Such patients are encouraging from the standpoint of the efficiency of present therapy. Clinical improvement paralleled the serologic results tabulated.

The accompanying chart shows a quantitative study by Kahn's method of another patient showing such rapid complete serologic reversal early in treatment with sulpharsphenamine. One should note that the Wassermann reaction reversed from a four plus to a negative in a single week (fifth week of treatment). The Kahn reading decreased from 240 to 4 units in the first four weeks of treatment and then continued to decline to a complete negative in an interval of about two and a half weeks. This patient, however, succumbed to a nonsyphilitic com-



Quantitative study of early complete reversal of results in serologic tests during treatment. The lower curve represents quantitative Kahn readings in Kahn units.

plication, intestinal intoxication, which was preceded by cellulitis of the neck.

Table 7 shows three different types of persistent positivity throughout months and years of treatment. The case of "Rose N." is an example of persistent strongly positive reactions in both tests during eighty-eight months of treatment and observation, being both a "Wassermann fast" and a "Kahn fast" case. The case of "Basile H." became and remained negative to complement fixation, but has frequently relapsed to weakly positive and strongly positive Kahn reactions. This is an example of a patient who remained "Kahn fast" despite the reversal of the Wassermann reaction, and affords the single instance of this type of persistent positivity in our experience. "Frank M." has

shown rapid, bizarre alternating fluctuations in both Wassermann and Kahn reactions throughout his treatment. This patient was previously referred to in the comment on table 2.

WASSERMANN AND KAHN TESTS IN EXAMINATION OF MOTHERS OF SYPHILITIC CHILDREN

The serologic reactions of the mothers of children under investigation for syphilis is usually of great diagnostic value. For this reason,

TABLE 7.—*Three Varieties of Persistent Positivity During Treatment*

Rose N., 17 Months			Basil H., 7 Months			Frank M., 1 Year		
Period Treated, Months	Wassermann	Kahn*	Period Treated, Months	Wassermann	Kahn	Period Treated, Months	Wassermann	Kahn
54	4	444	26	1	013	Before treatment	4	- - 0
66	4	444	30½	2	013	3 weeks later	4	- - 4
69	4	-44	34	0	000	4	4	4 4 4
72	4	444	36	0	113	8	0	0 0 0
76	4	444	38	0	234	10	4	0 0 0
84	4	444	44	0	001	11	0	0 2 2
88	4	444	48	0	001	12	0	0 0 1
88	4	444	52	0	123	14	0	2 2 3
			53	0	444	16½	0	0 0 1
			55	0	124			

* Quantitative procedure 20 reacting units.

TABLE 8.—*Comparative Sensitivity of Wassermann and Kahn Tests in Seventy-One Mothers of Syphilitic Offspring* †

Results in Agreement	Serums	Results in Disagreement	Serums	Comparative Sensitivity
Both tests +.....	41	Wassermann, +; Kahn, 0.....	3	3 cases favorable to Wassermann (sensitivity of 61.9%)
Both tests 0.....	4	Wassermann, +; Kahn, doubtful.	0	
Both tests doubtful	1	Wassermann, doubtful; Kahn, 0..	0	
Total.....	46	Wassermann, doubtful; Kahn, +.	4	22 cases favorable to Kahn (sensitivity 87.3%)
		Wassermann, 0; Kahn, doubtful..	1	
		Wassermann, 0; Kahn, +.....	17	
		Total.....	25	

† Seventy per cent of the mothers were examined earlier than six months postpartum.

the serologic results in examination of the 317 mothers of the patients discussed are included. These results have been classified wholly on the basis of the presence or the absence of syphilis in the offspring and not on the basis of evidence of syphilis in the mother herself. Thus, these mothers fall into two main groups: seventy-one mothers of syphilitic infants; 246 mothers of nonsyphilitic infants.

The serologic results of Wassermann and Kahn tests in examination of the seventy-one mothers of syphilitic infants are shown in Table 8. In forty-six serums, the two tests were in complete agreement, while in twenty-five serums the two tests were in disagreement. It is obvious that the two tests show a greater variance in the mothers of

TABLE 9.—*Detail of Examination of Twenty-Five Mothers of Syphilitic Offspring Whose Two Serological Tests Were in Disagreement*

(1) Wassermann, 0; Kahn, +; 17

Name	Age, Years	Pregnancy	Period Postpartum	Wassermann	Kahn	Child Wassermann	Child Kahn
Mrs. M.....	24	P*	1½ mo.	0	444	4	444
			9½ mo.	4	444	Treated	
Mrs. D.....	32	P	1½ mo.	0	444	4	444
			16½ mo.	4	444	Treated	
Mrs. B.....	22	P	2 mo.	0	444	4	444
			17 mo.	4	444	Not treated	
			21 mo.	4	444	Not treated	
Mrs. H.....	..	P	2 mo.	0	444	4	444
		Pregnant again	5 mo.	0	444		
Mrs. B.....	..	P	2½ mo.	0	444	4	444
Mrs. K.....	22	P	3 mo.	0	444	4	444
Mrs. Z.....	22	P	4 mo.	0	444	4	444
Mrs. S.....	24	P	7 mo.	0	444	4	444
Mrs. M.....	21	P	14 mo.	0	244	4	444
Mrs. L.....	..	P	4 mo.	0	444	4	-- 4

(2) Wassermann, 0; Kahn, doubtful; 1

Mrs. S.....	24	P	5½ mo.	0	012	Anticomplementary	222
			12½ mo.	1	134		

(3) Wassermann, doubtful; Kahn, +; 4

Mrs. H.....	20	P	2 mo.	2	444	4	444

Name	Age, Years	Pregnancy	Period Postpartum	Wassermann	Kahn	Child Wassermann	Child Kahn
Mrs. S.....	19	M 2	1 mo.	1	444	4	444
			5 wk.	3	444		
Mrs. M.....	27	M 2	2 mo.	2	444	4	444
Mrs. W.....	36	M 2	4 mo.	1	122	4	411

(4) Wassermann, +; Kahn, 0; 3

Mrs. B.....	19	P	2 mo.	4	000	D.F.S.‡	
Mrs. T.....	32	P	24 mo.	4	000	4	011

Name	Age, Years	Pregnancy	Period Postpartum	Wassermann	Kahn	Child Wassermann	Child Kahn
Mrs. N.....	28	M 4	2 mo.	4	000	4	444

The superiority of the Kahn test is indicated. The high incidence of primiparae should be noted. The average age of the mothers was 27 years (the same both for those whose two tests were in agreement and for those whose two tests were in disagreement)

* P = primiparous.

† M = multiparous.

‡ D.F.S. = definite familial syphilis.

syphilitic infants, 35.3 per cent, than they do in the syphilitic infants themselves, 13.4 per cent (table 3). An analysis of these twenty-five discrepancies shows that twenty-two are favorable to the Kahn test (the positivity of the Kahn test is greater) and three favorable to the Wassermann test. It should be noted that these are not minor differences, as seventeen show an absolute disagreement, favorable to the Kahn test, and three show an absolute disagreement, favorable to the Wassermann test. The details of the examination of the mothers whose two tests were in disagreement are listed in table 9. This group was further analyzed to determine whether the age of the mother, the number of pregnancies and the length of the postpartum period were factors operating in the production of serologic disagreement. These results are tabulated in table 10. It would seem unwise to generalize on a group of this size, but in these twenty-five cases the Wassermann-

TABLE 10.—*Influence of Age of Mother, Number of Pregnancies and Period Post Partum in Producing Serologic Discrepancies*

Groups	Average Age of Mother, Years	Number of Pregnancies		Period Postpartum	
		Primipara, per Cent	Multipara, per Cent	Less Than 1 Year, per Cent	More Than 1 Year, per Cent
Entire group	27	32	68	76	24
Group whose two tests were in agreement	27	28	72	72	28
Group whose two tests were in disagreement (Wassermann, 0; Kahn, +)	27	42	58	88	12
	No effect	Moderate increase in primiparae		In 70 per cent, less than 3 months	

negative, Kahn-positive combination is found more frequently in primiparous women early in the postpartum period than in multiparas, later postpartum. A summary of these results gives a Kahn sensitivity of 87.3 per cent as compared with a Wassermann sensitivity of 61.9 per cent. Neither test was as sensitive in the case of the mothers of syphilitic offspring as in the case of the offspring themselves. There is an apparent advantage here of 25.4 per cent for the sensitivity of the Kahn reaction.

The serologic results in the examination of 246 mothers of non-syphilitic infants are shown in table 11. This table demonstrates the specificity of the two tests in the case of the mothers whose offspring were free from syphilis. Two hundred thirty-one serums showed the two tests in agreement, and there were fifteen discrepancies. The group in agreement shows five mothers positive in both tests and probably syphilitic, whose children escaped the infection. Of the fifteen discrepancies, eleven showed the Kahn test more positive than the Wassermann. In serums of ten mothers whose children were free from syphilis, the two tests were in absolute disagreement, the Kahn test

indicating eight of them as positive. One might think that the Kahn test was oversensitive and was giving false positive reactions in this group, were it not for the fact that five of the eight Wassermann-negative, Kahn-positive mothers were known to have syphilis and had received antisyphilitic treatment ante partum. The details of the ten absolute discrepancies in table 11 are given in table 12. A summation of the specificity in mothers of nonsyphilitic offspring shows: Wassermann, 95.5 per cent; Kahn, 94.7 per cent.

TABLE 11.—*Comparative Specificity of the Wassermann and Kahn Tests in Two Hundred and Forty-Six Mothers of Nonsyphilitic Offspring*

Results in Agreement	Serums	Results in Disagreement	Serums
Wassermann, +; Kahn, +	5	Wassermann, +; Kahn, 0	2
Wassermann, doubtful; Kohn, doubtful	2	Wassermann, doubtful; Kahn, 0	2
Wassermann, 0; Kahn, 0	224	Wassermann, 0; Kahn, doubtful	3
		Wassermann, 0; Kahn, +	8
Total	231	Total	15
		(Five were treated during pregnancy)	

TABLE 12.—*Detail of Examination of Ten Mothers of Nonsyphilitic Offspring Whose Parallel Tests Were in Absolute Disagreement*

Name	Age, Years	Period Postpartum	Wassermann	Kahn	Remarks
Wassermann, +; Kahn, 0 = 2 Mothers					
Mrs. W.	28	5 yr.	4	000	Infant negative; no familial syphilis
Mrs. A.	?	14 mo.	4	000	Infant negative; no familial syphilis
Wassermann, 0; Kahn, + = 8 Mothers *					
Mrs. A.	25	12 wk.	0	444	Infant negative
		22 wk.	4	444	Infant negative
		38 wk.	4	444	Treatment during pregnancy
Mrs. R.	28	2½ yr.	0	144	Infant negative; treated during pregnancy
Mrs. C.	28	6 wk.	0	234	Infant negative; treated during pregnancy
Mrs. P.	27	4 mo.	0	444	Infant negative; no treatment; no familial syphilis
Mrs. K.	29	6 mo.	0	123	Infant negative; no familial syphilis
Mrs. D.	27	7 mo.	0	444	Infant negative
		8 mo.	0	133	Treatment during pregnancy
Mrs. S.	32	1 yr.	0	444	Infant negative; no treatment
Mrs. D.	32	3 mo.	0	113	Infant negative; mother treated

* Five were treated during pregnancy.

The tendency of pregnancy to weaken complement fixation and give negative Wassermann reactions in syphilitic pregnant women has been widely observed.[15] This phenomenon has no doubt frequently caused failure of diagnosis of syphilis during pregnancy and has postponed treatment during pregnancy, with the result of infection of the fetus. A serologic test which would detect syphilis in this negative phase of the Wassermann reaction would be a valuable diagnostic aid and a factor in the prevention of infantile syphilis. We have had no

15. Stokes, J. H.: Modern Clinical Syphilology, Philadelphia and London, W. B. Saunders & Company, 1926, p. 1007.

experience with serums of antepartum women, but our results with syphilitic mothers early post partum indicates that the Kahn precipitation test is considerably more sensitive than the Wassermann test at this time. Hinton's [16] results with the serums of pregnant women suggest that the Kahn test is also more sensitive ante partum.

A condensed summary of results appears in table 13.

CONCLUSIONS

1. The Kahn test is a highly sensitive and highly specific serologic test in infancy and early childhood. The reaction closely parallels the Wassermann reaction, but is not identical with it.

2. Positive Wassermann reactions are more easily reversed by antisyphilitic therapy than are positive Kahn reactions.

TABLE 13.—*Condensed Summary of Results*

1,185 Infants and Young Children		43 Patients Under Treatment for Syphilis		316 Postpartum Mothers	
Sensitivity in 104 Who Were Syphilitic				Sensitivity in 71 Mothers of Syphilitic Offspring	
Wassermann +.....	91.3%	Both tests in agreement...	79.9%	Wassermann +.....	61.9%
Kahn +.....	93.2%	Both tests in disagreement	20.1%	Kahn +.....	87.3%
Specificity in 1,081 Who Were Not Syphilitic		Favorable to Wassermann.....	3.3%	Advantage for Kahn test.....	25.4%
Wassermann 0.....	98.8%	Favorable to Kahn.....	16.8%		
Kahn 0.....	98.1%	Advantage for Kahn test	13.5%	Specificity in 246 Mothers of Nonsyphilitic Offspring	
				Wassermann 0.....	95.5%
				Kahn 0.....	94.7%

3. The Kahn test is considerably more sensitive than the Wassermann test in mothers of syphilitic offspring. It is highly specific in mothers of nonsyphilitic offspring and equals the Wassermann test in this regard.

4. The Kahn test has decided advantages in simplicity as a laboratory procedure.

5. The Kahn test performed alone would have given slightly more reliable information than the Wassermann test performed alone on this group of patients.

6. The performance of both complement fixation and precipitation tests simultaneously on identical serums gives more information than either test alone and affords a duel control in serologic diagnosis, which seems desirable.

The Babies' Hospital, 167th Street and Broadway, New York.

16. Hinton, W. A.: Kahn and Wassermann Reactions, Boston M. & S. J. **194**:5, 1926.

THE RELATIONS OF WEIGHT, HEIGHT AND AGE IN NEGRO CHILDREN*

L. T. ROYSTER, M.D.
AND
C. N. HULVEY, M.S., L.L.B.
UNIVERSITY OF VIRGINIA, VA.

Weight in relation to height furnishes, at present, the best single criterion of the state of a child's nutrition. The relation of weight for age has been entirely abandoned, except for the first three years of life, when it is almost as accurate as weight for height. Such a criterion is merely a guide, however, based on an average from a large number of children; how far a child may vary from the average and still not be malnourished must be determined ultimately by the judgment of the individual examiner, such judgment to be based on extensive experience and familiarity with both normal and malnourished children.

The studies of Baldwin and Wood and the tables computed from their studies have been the accepted guide in routine examinations for a long time. These studies are based on the measurements and weights of a large number of unselected school children, and it is presumed that a certain amount of racial admixture was inevitable in so large a number of children. Few studies have been made on negro children alone; hence the curves plotted by Baldwin and Wood have been the accepted guide for the negroes as well as for the whites. Among clinics everywhere, but especially in the Southern states, the question has naturally arisen whether these standards are applicable to negro children; in fact, a number of clinicians have stated that it was their impression that negro children averaged heavier for their height than did white children. The authors therefore made the present study with a view of determining these facts.

About 10,000 children in all were measured and weighed, but on calculating averages, we found that there were too few represented at certain points of the scale, particularly at each end of the age or the height records to furnish reliable figures; the data on a little over 1,000 were therefore discarded, and the tables, as given, were compiled from data on 8,876 children, comprising 4,595 girls and 4,281 boys. A majority of the children were from the public schools of Richmond, Va., a small number from the public schools of Charlottesville, Va.,

* Submitted for publication, June 5, 1929.

* From the Department of Pediatrics, University of Virginia.

and something less than a hundred from Greene County, Va. The study, therefore, is one of urban children, the small number from the county schools having no material effect on the average. The data from all sources were thrown into the general average. All children were

TABLE 1.—*A Comparison of the Average Weight for Height for Age of Negro Boys with the Corresponding Data for White Boys, as Given in the Table of Baldwin and Wood*

	White Boys, Age												
Height	6	7	8	9	10	11	12	13	14	15	16	17	18
45	46	46	46	46	...	...	...	...	...	...	...	...	...
46	48	48	48	48	...	...	...	...	...	...	...	...	...
47	50	50	50	50	50	...	...	...	...	...	...	...	...
48	52	53	53	53	53	...	...	...	...	...	...	...	...
49	55	55	55	55	55	55	...	...	...	...	...	...	...
50	57	58	58	58	58	58	58	...	...	...	...	...	...
51	..	61	61	61	61	61	61	...	...	...	...	...	...
52	..	63	64	64	64	64	64	64	...	...	...	...	...
53	..	66	67	67	67	67	68	68	...	...	...	...	...
54	..	..	70	70	70	70	71	71	72	...	...	...	...
55	..	.	72	72	73	73	74	74	74	...	...	...	...
56	..	..	75	76	77	77	77	78	78	80	...	...	...
57	..	..	..	79	80	81	81	82	83	83	...	...	...
58	..	..	..	83	84	84	85	85	86	87	...	...	...
59	..	..	..	..	87	88	89	89	90	90	90	...	...
60	..	..	..	..	91	92	92	93	94	95	96	...	...
61	..	..	..	..	95	96	97	99	100	103	106	...	...
62	..	..	..	..	100	101	102	103	104	107	111	116	...
63	..	..	..	..	105	106	107	108	110	113	118	122	127
64	..	..	..	..	...	109	111	113	115	117	121	126	130
65	..	..	..	..	...	114	117	118	120	122	127	131	134
66	..	..	..	..	...	...	119	122	125	128	132	136	139
67	..	..	..	..	...	...	124	128	130	134	136	139	142
		Colored Boys, Age											
Height	6	7	8	9	10	11	12	13	14	15	16	17	18
45	46	46	46	46	...	...	...	...	...	...	...	...	...
46	48	48	48	48	...	...	...	...	...	...	...	...	...
47	50	50	50	50	50	...	...	...	...	...	...	...	...
48	52	53	53	53	53	...	...	...	...	...	...	...	...
49	55	55	55	55	55	...	...	...	...	...	...	...	...
50	57	58	58	58	58	58	58	...	...	...	...	...	...
51	..	61	61	61	61	61	61	...	...	...	...	...	...
52	..	63	64	64	64	64	64	64	...	...	...	...	...
53	..	..	67	67	67	67	68	68	...	...	...	...	...
54	..	..	70	70	70	70	71	71	...	...	...	...	...
55	..	..	72	72	73	73	74	74	...	...	...	...	...
56	..	..	..	76	77	77	77	78	78	...	...	...	...
57	..	..	..	79	80	81	81	82	83	83	...	...	...
58	..	..	..	..	84	84	85	85	86	87	...	...	...
59	..	..	..	..	87	88	89	89	90	90	...	...	...
60	..	..	..	..	91	92	92	93	94	95	96	...	...
61	..	..	..	..	..	95	96	97	99	100	...	...	...
62	..	..	..	..	..	100	101	102	103	104	...	...	...
63	..	..	..	..	..	...	106	107	108	110	...	...	...
64	..	..	..	..	..	...	109	111	113	115	117	...	...
65	..	..	..	..	..	...	114	117	118	120	122	...	...
66	..	..	..	..	..	...	...	...	122	125	...	...	...
67	..	..	..	..	..	...	...	...	128	...	...	...	...

lightly clothed, the wraps of the girls and the coats of the boys having been removed, and the heights were measured without shoes. All weights and measurements were those recorded in the course of the routine school clinics and inspections. The children of Charlottesville and Greene County were measured and weighed under the supervision of one of us, while those of Richmond were under the supervision

of Dr. N. Thomas Ennett, medical director of the Richmond School System, who allowed us to use his records. No attempt was made to record shades of color.

The data were first arranged to show the relation of weight to height for age, and compared with the familiar tables of Baldwin and

TABLE 2.—*A Comparison of the Average Weight for Height for Age of Negro Girls with the Corresponding Data in the Baldwin-Wood Table for White Girls*

	White Girls, Age													
Height	5	6	7	8	9	10	11	12	13	14	15	16	17	18
45	45	45	45	45	45	...	...	...	...	...	...	...	...	...
46	47	47	47	48	48	...	...	...	...	...	...	...	...	...
47	49	50	50	50	50	50	...	...	...	...	...	...	...	...
48	..	52	52	52	52	53	53	...	...	...	...	...	...	...
49	..	54	54	55	55	56	56	...	...	...	...	...	...	...
50	..	56	56	57	58	59	61	62	...	...	...	...	...	...
51	..	..	59	60	61	61	63	65	...	...	...	...	...	...
52	..	..	63	64	64	64	65	67	...	...	...	...	...	...
53	..	..	66	67	67	68	68	69	71	...	...	...	...	...
54	..	..	..	69	70	70	71	71	73	...	...	...	...	...
55	..	..	..	72	74	74	74	75	77	78	...	...	...	...
56	..	..	..	..	76	78	78	79	81	83	...	...	...	...
57	..	..	..	..	80	82	82	82	84	88	92	...	...	...
58	..	..	..	..	..	84	86	86	88	93	96	101	...	...
59	..	..	..	..	..	87	90	90	92	96	100	103	104	...
60	..	..	..	..	..	91	95	95	97	101	105	108	109	111
61	..	..	..	..	..	..	99	100	101	105	108	112	113	116
62	..	..	..	..	..	..	104	105	106	109	113	115	117	118
63	..	..	..	..	..	..	...	110	110	112	116	117	119	120
64	..	..	..	..	..	..	...	114	115	117	119	120	122	123
65	..	..	..	..	..	..	...	118	120	121	122	123	125	126

	Colored Girls, Age													
Height	5	6	7	8	9	10	11	12	13	14	15	16	17	18
45	45	45	45	45	45	...	...	...	...	...	...	...	...	...
46	..	47	47	48	48	...	...	...	...	...	...	...	...	...
47	..	50	50	50	50	50	...	...	...	...	...	...	...	...
48	..	52	52	52	52	53	...	...	...	...	...	...	...	...
49	..	54	54	55	55	56	56	...	...	...	...	...	...	...
50	..	56	56	57	58	59	61	62	...	...	...	...	...	...
51	..	59	59	60	61	61	63	65	...	...	...	...	...	...
52	..	..	63	64	64	64	65	67	...	...	...	...	...	...
53	..	..	66	67	67	68	68	69	...	...	...	...	...	...
54	..	..	67	69	70	70	71	71	73	...	...	...	...	...
55	..	..	..	72	74	74	74	75	77	...	...	...	...	...
56	..	..	..	..	76	78	78	79	81	83	...	...	...	...
57	..	..	..	..	80	82	82	82	84	88	...	...	...	...
58	..	..	..	..	..	84	86	86	88	93	96	...	...	...
59	..	..	..	..	..	87	90	90	92	96	100	...	...	...
60	..	..	..	..	..	..	95	95	97	101	105	108	...	...
61	..	..	..	..	..	..	99	100	101	105	108	112	...	...
62	..	..	..	..	..	..	104	105	106	109	110	115	117	...
63	..	..	..	..	..	..	...	110	110	112	...	...	...	...
64	..	..	..	..	..	..	...	...	115	117	119	120	...	...
65	..	..	..	..	..	..	...	...	120	121	122	...	...	...

Wood.[1] Table 1, for boys, shows a practical identity of weights for height for age of colored boys and white boys measuring from 45 to 60 inches (114.30 to 152.4 cm.) inclusive. The negro boys from 61 to 67 inches (155-170.2 cm.) in height are from 1 to 3 pounds (0.5 to 1.4 Kg.) lighter for height at each age, except that at 65 inches

1. The tables were those published and distributed, for general use, by the American Child Health Association.

(165.1 cm.) of height, at 16 years of age, the negro boy is 5 pounds (2.3 Kg.) lighter than the white boy of like height and age. Table 2, for girls, shows the weights for height of the colored girls and the white girls to be practically identical throughout.

The data on the colored children were then arranged so as to show weight for height only, without regard to the age incidence (table 3). The tables used for comparison were those compiled for white children by the National Health Council, based on the tables of Baldwin and Wood and adopted and published by the Nutrition Clinics for Delicate Children, of Boston (Emerson).

TABLE 3.—*A Comparison of the Average Weight for Height of Negro Children with the Corresponding Data for White Children, as Given by Baldwin and Wood*

Height	Negro Boys' Average Weight	White Boys' Average Weight	Variation	Height	Negro Girls' Average Weight	White Girls' Average Weight	Variation
45	46	46	0	45	45	45	0
46	48	48	0	46	47	47	0
47	50	50	0	47	50	50	0
48	53	53	0	48	52	52	0
49	55	55	0	49	55	55	0
50	58	58	0	50	58	58	0
51	61	61	0	51	61	61	0
52	64	64	0	52	64	64	0
53	67	68	−1	53	68	68	0
54	70	71	−1	54	70	71	−1
55	73	74	−1	55	74	75	−1
56	77	78	−1	56	78	79	−1
57	81	82	−1	57	82	84	−2
58	85	85	0	58	87	89	−2
59	89	89	0	59	92	95	−3
60	93	94	−1	60	100	101	−1
61	97	99	−2	61	105	108	−3
62	102	104	−2	62	109	114	−5
63	107	111	−4	63	112	118	−6
64	113	117	−4	64	118	121	−3
65	118	123	−5	65	119	125	−6

These figures are averaged in even inches and pounds, no fractions being used. With the data thus arranged, some slight differences from the former comparison are shown in the middle scale, and marked differences in the upper scale. It may be seen that the average weight according to height is the same for negro children as for white children until the height of 52 inches (132.1 cm.) is reached; after this, negro children weigh less for their height than do white children, and the variation constantly becomes greater thereafter, being 5 pounds (2.3 Kg.) for boys and 6 pounds (2.7 Kg.) for girls at 65 inches (165.1 cm.).

A comparison of the height weight curve for negro boys with that for negro girls is shown in chart 1. This comparison shows that at heights of from 45 to 49 inches (114.30 to 124.5 cm.) negro girls weigh slightly less than negro boys of the same heights, except that

at a height of 47 inches (119.38 cm.) they weigh the same. At heights of from 49 to 52 inches (124.5 to 132.1 cm.), the weights for boys and those for girls are identical. At heights of from 52 to 65 inches (132.1 to 165.1 cm.) inclusive, the girls are constantly heavier than the boys, with the exception that at the height of 54 inches (137.2 cm.) their weights are identical. These differences vary from a few ounces

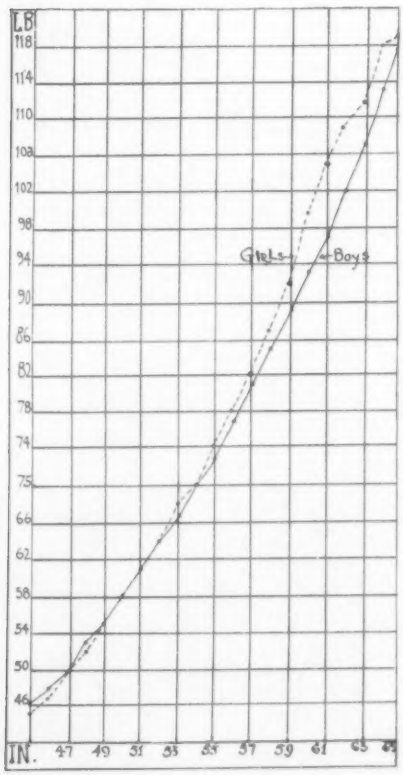


Chart 1

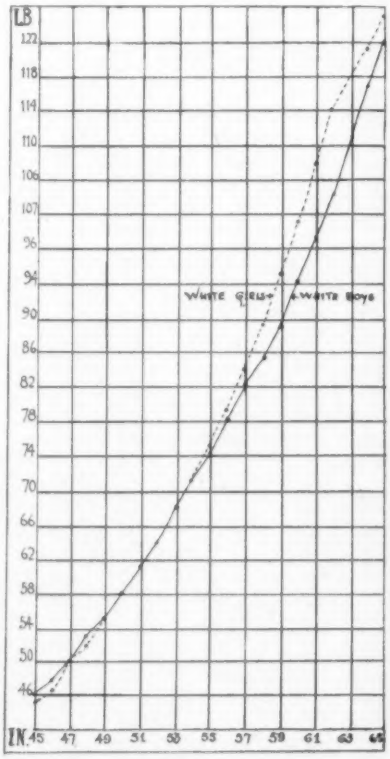


Chart 2

Chart 1.—A comparison of the height for weight curve for negro boys with that for negro girls.

Chart 2.—A comparison of the height for weight curve for white boys with that for white girls, from the studies of Baldwin and Wood.

to 8 pounds (3.6 Kg.) (at the height of 61 inches [155 cm.]). The two sexes weigh approximately the same at the height of 65 inches (165.1 cm.) (15 years).

Chart 2 shows a comparison of the weight height curve for white boys with that for white girls as computed from the studies of Baldwin

and Wood. The chart presents approximately the same variation as do the studies of colored children.

A comparison was then made between our observations in negro children and those of Benedict and Talbot [2] in public school children. The comparison shows the negro children whom we studied to be slightly heavier than the children studied by Benedict and Talbot.

Mustard and Waring,[3] working in the Child Health Demonstration of Rutherford County, Tenn., reported that "colored children of school

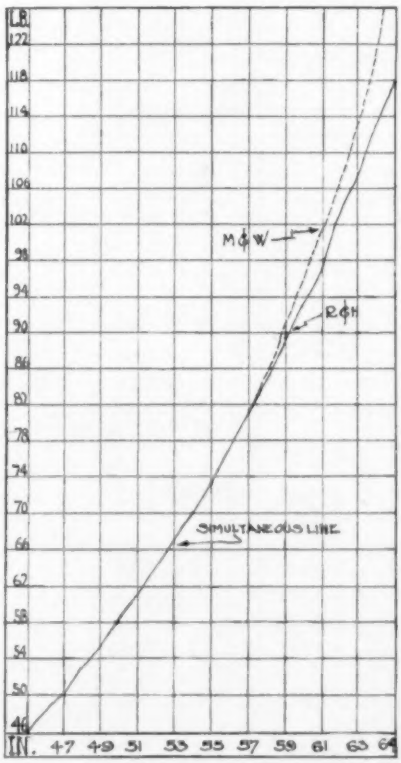


Chart 3

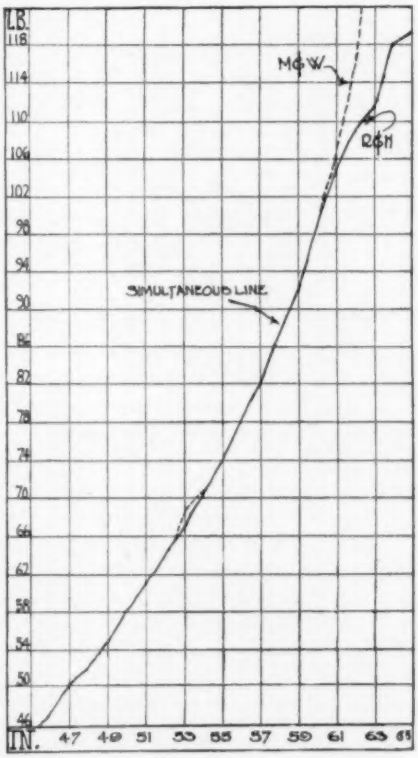


Chart 4

Chart 3.—A comparison of the height for weight ratio curve for Royster and Hulvey's colored boys with that for Mustard and Waring's colored boys.

Chart 4.—A comparison of the height for weight ratio curve for Royster and Hulvey's colored girls with that for Mustard and Waring's colored girls.

age were found uniformly heavier than white children of the same age, and taller than white children until toward the end of adolescence."

2. Benedict and Talbot: Metabolism and Growth from Birth to Puberty, Washington, D. C., Carnegie Institution of Washington, 1921, pp. 45 and 46.

3. Mustard, H. S., and Waring, J. I.: Heights and Weights of Colored School Children, Am. J. Pub. Health **16**:1017 (Oct.) 1926.

Charts 3 and 4 show the weight for height of the colored boys and girls whom we studied compared with the weight for height of those studied by Mustard and Waring. Mustard and Waring studied a total of 1,650 colored children; our studies were made on a much larger number.

Studies were then made of the relation of height to age. The data on the height for age of white boys and girls were taken from the tables of Baldwin and Wood and the corresponding data for the

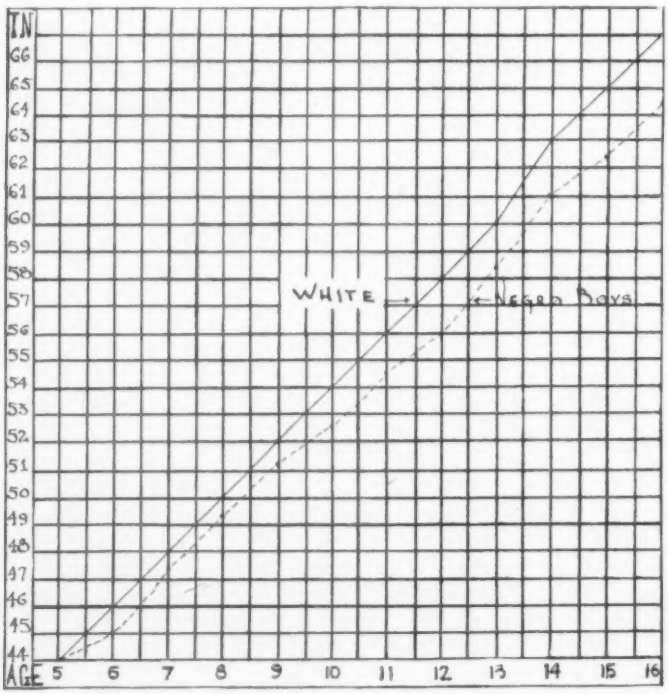


Chart 5.—A comparison of the height for age curve for negro boys with that for white boys.

negro boys and girls were compared with them. The data for boys show that there is no variation at 5 years, but that from the 6th to the 16th years inclusive, there is a constant minus variation for negro boys, ranging from one-half inch (1.27 cm.) to 2.8 inches (7 cm.) with minor periodic variations. These observations are shown graphically in chart 5.

The relation of height to age for girls shows striking differences. At the 6th and 7th years, negro girls show a plus variation over white girls of a little less than one-half inch. Between the 7th and

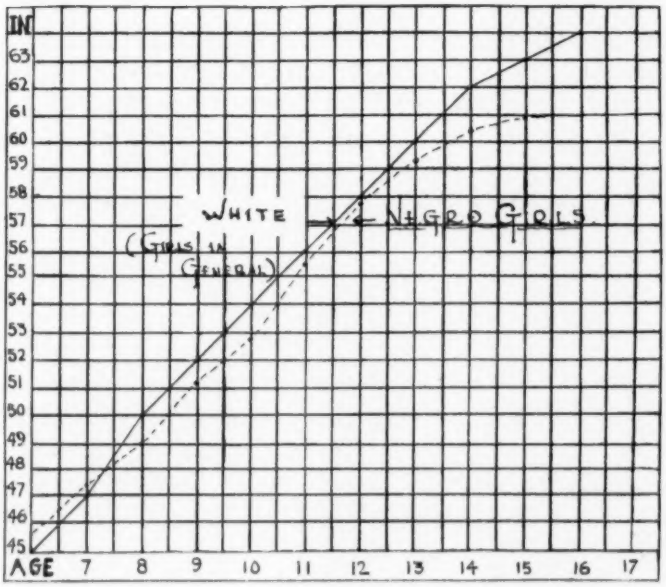


Chart 6.—A comparison of the height for age curve for negro girls with that for white girls.

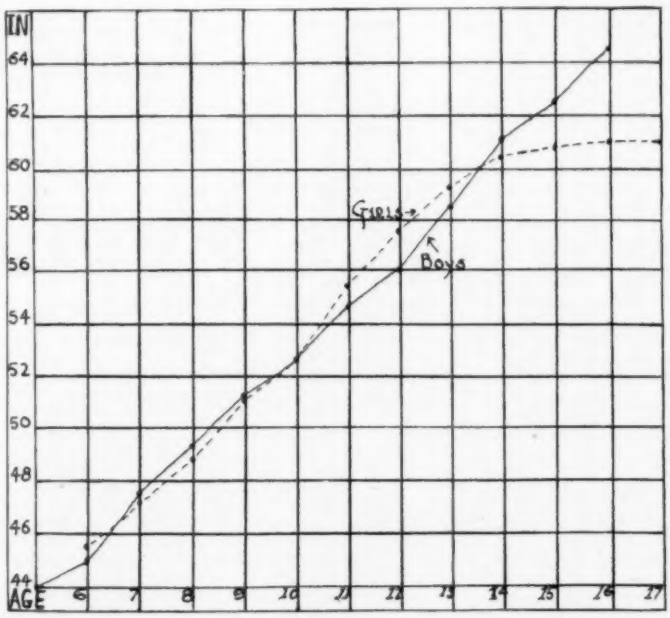


Chart 7.—A comparison of the height for age curve for negro boys with that for negro girls.

8th years, the lines cross, and from then on the height of negro girls is constantly below that of white girls, the minus variation ranging from 1 to 3 inches (2.5 to 7.6 cm.). The maximal growth of white girls is shown at the 16th year; at this time they are 64 inches (162.6 cm.) tall. The maximal growth of the negro girl is reached at 15½ years; at this time she is 61 inches (155 cm.) tall. These data are shown graphically in chart 6.

TABLE 4.—*A Comparison of the Average Height for Age of White Boys (Baldwin and Wood) with That of Colored Boys (Royster and Hulvey)*

Number of Boys Studied	Age, Nearest Birthday	Height, Ins.	Weight, Lbs.	Variation
15	5	44.00	44	0.00
207	6	45.00	46	—1.00
666	7	47.46	48	— .54
772	8	49.30	50	— .70
589	9	50.80	52	—1.20
499	10	52.60	54	—1.40
431	11	54.60	56	—1.40
439	12	56.00	58	—2.00
361	13	58.50	60	—1.50
223	14	61.00	63	—2.00
64	15	62.20	65	—2.80
17	16	64.30	67	—2.70

TABLE 5.—*A Comparison of the Average Height for Age of White Girls with that of Negro Girls*

Number of Girls Studied	Age, Nearest Birthday	Height, Ins.	Weight, Lbs.	Variation
515	6	45.57	45	+ .43
674	7	47.30	47	+ .30
775	8	49.00	50	—1.00
526	9	51.12	52	— .88
487	10	52.80	54	—1.20
438	11	55.50	56	— .50
475	12	57.80	58	— .20
384	13	59.33	60	— .67
224	14	60.37	62	—1.63
72	15	60.88	63	—2.12
25	16	61.00	64	—3.00

A comparison of the age height ratio in negro boys with that in negro girls (chart 7) shows three points at which the lines cross. Up to 6½ years, the negro girl is slightly taller than the negro boy. At from 6½ to 10 years, she is slightly shorter. At from 10 to 13½ years the negro girl is definitely taller than the negro boy, being nearly 2 inches (5 cm.) taller at the 12th year. Between the 13th and the 14th years, the lines cross for the last time at 60 inches (152.4 cm.) and from this time on there is a constantly increasing variation until at 16 years the girls are found to be 3½ inches (8.87 cm.) shorter than the boys. These variations are shown graphically in chart 7.

Case Reports

SPLENOMEGALY
PORTAL PHLEBOSCLEROSIS *

SAMPSON J. WILSON, M.D.
AND
MAX LEDERER, M.D.
BROOKLYN

Among the cases of splenomegaly encountered in pediatric practice there are many that closely resemble Banti's disease in their clinical and laboratory aspects. These lead to errors in diagnosis. Cases originally reported as Banti's disease are proved, on further study or at autopsy, to be cases of splenomegaly due to some obstruction in the portal circulation, with a coexisting thrombosis or sclerosis of the splenic or portal veins. Thus, Marie Haenelt,[1] in reporting a case of this kind, mentioned eight cases found in the literature which were designated as Banti's disease but which at autopsy were proved to be cases of either portal or splenic phlebosclerosis. This condition is rather rare in children but is of great importance to the pediatrician, for not infrequently some of its causative factors and clinical manifestations make their first appearance during infancy. We are therefore reporting a case of this type in an infant aged 16 months.

REPORT OF CASE

History.—A boy, aged 1 year and 4 months, of Jewish parentage, was admitted to the Jewish Hospital of Brooklyn, on Feb. 2, 1928, with the complaint of having vomited a large amount of blood and clots of blood the day before. The father was 32 years of age and had suffered from cardiac disease for nine years. The mother, aged 27, was well. She had had three pregnancies. The first child, a boy, born eight years before, died of cardiac disease at 6 years of age. The second child, a girl, born four years before the admission of the patient, died three days after birth, from unknown causes. The patient was born at full term, the delivery being spontaneous with chloroform anesthesia. He was asphyxiated at birth and on the second day, because of the persisting cyanosis, he was given paternal blood intramuscularly. He was icteric during the first five days. There was no history of eruptions, umbilical infection or acute illness. The patient had always been pale. The abdomen had been round and protuberant since birth.

* Submitted for publication, July 10, 1929.

* From the Departments of Pediatrics and Pathology, Jewish Hospital of Brooklyn.

1. Haenelt, Marie: Med. Klin. **24**:622 (April 20) 1928.

The patient was breast fed for four months, and was then fed with modifications of whole milk and dextrimaltose. Whole milk was given at 6 months of age; cod liver oil, at 3 months. The appetite had been poor for seven months, but there had been no nausea or vomiting until the present illness. Occasionally the patient was constipated, more so for the three weeks preceding admission, necessitating frequent enemas. Two months before admission, a streak of fresh blood was found in the stool. The developmental history was normal. The patient held his head up at 4 months of age, and sat up at 4½ months; the first tooth appeared at 8 months, and walked at 11 months.

The night before admission to the hospital, after one slight hacking cough, the patient vomited about a pint of dark brown blood containing many large clots and pieces of undigested food. He turned pale for a short time; then he became active again, and his color improved. No bleeding was noted at that time.

Physical Examination.—The general appearance was that of a well developed and well nourished infant of 15 months with marked pallor, appearing acutely ill but having no pain. The temperature was 99.6 F. There was no bleeding from the visible mucous membranes. The pharynx was slightly injected, and the tonsils were slightly enlarged. The anterior and posterior cervical nodes were

Composition of the Blood

Date, 1928	Hemoglobin, per Cent	Leukocytes	Polymorpho-nuclears	Lymphocytes	Large Mono-nuclears and Transitionals
2/ 2	25	12,600	76	20	4.0
2/ 5	40	4,800	69	21	10.0
2/ 8	34	10,250	50	45	3.5
2/13	55	11,600	66	30	3.5

palpable. A soft blowing systolic murmur was heard over the mitral area; otherwise the heart was normal. The abdomen was markedly distended. The liver was palpable one and one-half fingerbreadths below the costal margin and felt firm. The spleen was felt two fingerbreadths below the costal margin. The edge was firm and sharp. The other viscera were not palpable. A few purpuric spots were observed over the anterior and lateral surfaces of the lower extremities.

Clinical Course.—During the first week, the temperature fluctuated between 100 and 102 F., owing to a nasopharyngitis. During the second week it remained under 100 F. The patient received a transfusion of 160 cc. of unmodified blood on the second day after admission. A few days later, the spleen became slightly larger (three fingerbreadths below the costal margin). A second transfusion was given one week later. During the last few days in the hospital, the patient was comfortable and playful and had regular bowel movements.

The urine was normal on all occasions, but it showed urobilin in increased amounts. The blood platelets numbered 170,000 per cubic millimeter. The clot retraction time was one hour and forty-five minutes. The erythrocytes showed anisocytosis, poikilocytosis, oligochromia and an occasional macrocyte. The bleeding time was six minutes, the coagulation time seven minutes. The guaiac test of the stool gave positive results on February 6 and 10. The injection of 1 mg. of tuberculin gave negative results. The Wassermann reaction was negative. The direct van den Bergh test gave negative results; the indirect, 1 unit. The icterus index was 6.

The patient was discharged as improved after two weeks in the hospital with a diagnosis of possible purpura hemorrhagica.

He was readmitted one year later with the following intervening history: Four months after he left the hospital, an increasing pallor developed, and a transfusion was given at the Babies' Hospital. For three months following this transfusion, his abdomen grew progressively larger. The patient was admitted to the Mount Sinai Hospital, where he had a profuse hemorrhage from both mouth and rectum, which necessitated a second transfusion. His condition remained good for five weeks. Then he had a mild gastric hemorrhage. About

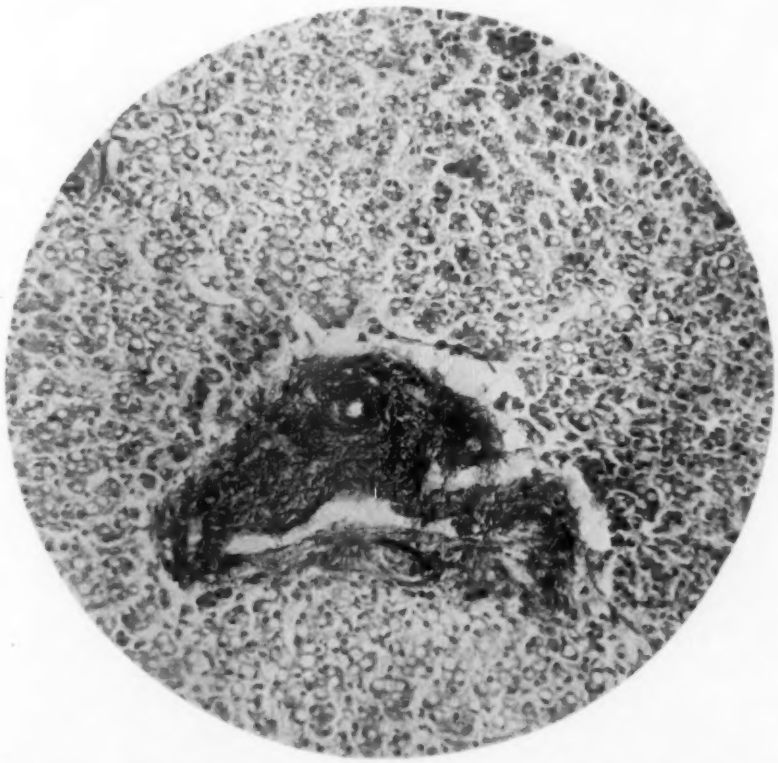


Fig. 1.—Fatty degeneration of the liver. Note the absence of interstitial changes and normal vessels in the portal areas. Hematoxylin-eosin; reduced from a magnification of × 100.

nine days before the second admission a temperature of 105 F. developed; during the next four days the temperature fluctuated between 101 and 105 F. This was followed by tarry stools for two days and a bright red, bloody vomitus. A transfusion was given, with no recurrence of bleeding. The child appeared to be very ill, and his condition did not improve. The temperature continued high, and the patient was in semicoma when readmitted.

Physical examination showed a pale, listless boy, aged 2 years, breathing superficially. Both fontanels were closed. Both ear drums were injected and

bulging. The tongue was dry and coated; tonsillar tissue was present, and the pharynx was injected. The cervical and inguinal nodes were palpable. A systolic apical murmur was heard, which was transmitted to the pulmonic area. A few crepitant râles were heard at the base of the right lung posteriorly and in the right axilla. The abdomen was markedly distended; the superficial veins were prominent, and no fluid wave could be made out. The liver was palpable 1 cm. and the spleen 4 cm. below the costal margin. The extremities showed evidence of dehydration. There was a healing wound in the right antecubital

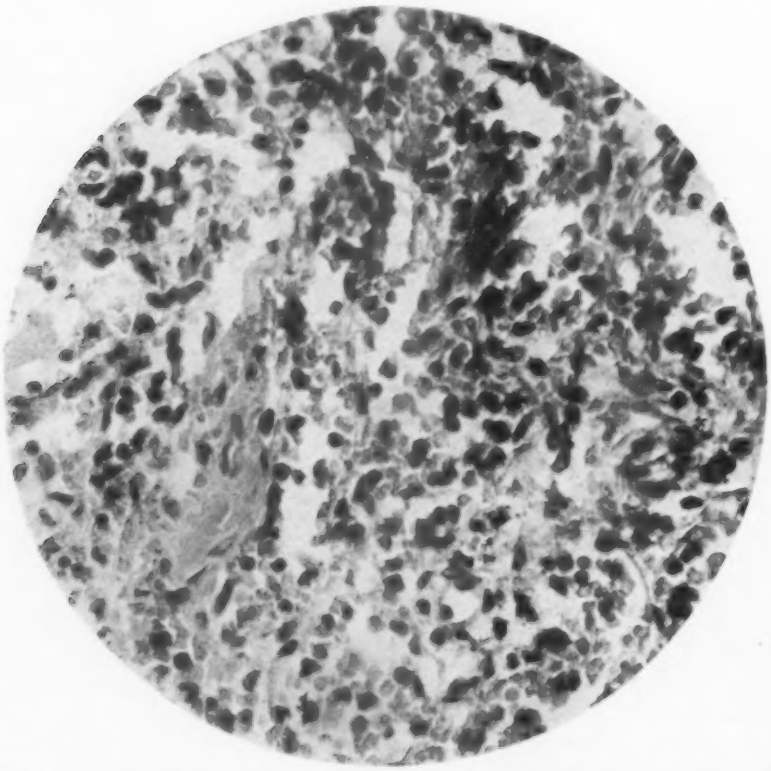


Fig. 2.—Interstitial changes in the spleen, with dilatation of the sinuses, endothelial hyperplasia and fibrous changes of the sinus walls. Hematoxylin-eosin; reduced from a magnification of × 400.

fossa (caused by transfusion) and a healed scar in the left. There were no abnormal neurologic manifestations.

Laboratory Data: Examination of the urine showed nothing abnormal. The blood contained: hemoglobin, 54 per cent; erythrocytes, 2,900,000; leukocytes, 31,600; neutrophils, 73; lymphocytes, 10 and mononuclears, 7 per cent, and platelets, 106,000. The bleeding time was two and one-half minutes and the coagulation time three and one-half minutes. Examination of the stool showed a markedly positive reaction to the guaiac test, but no ova or parasites. Three days

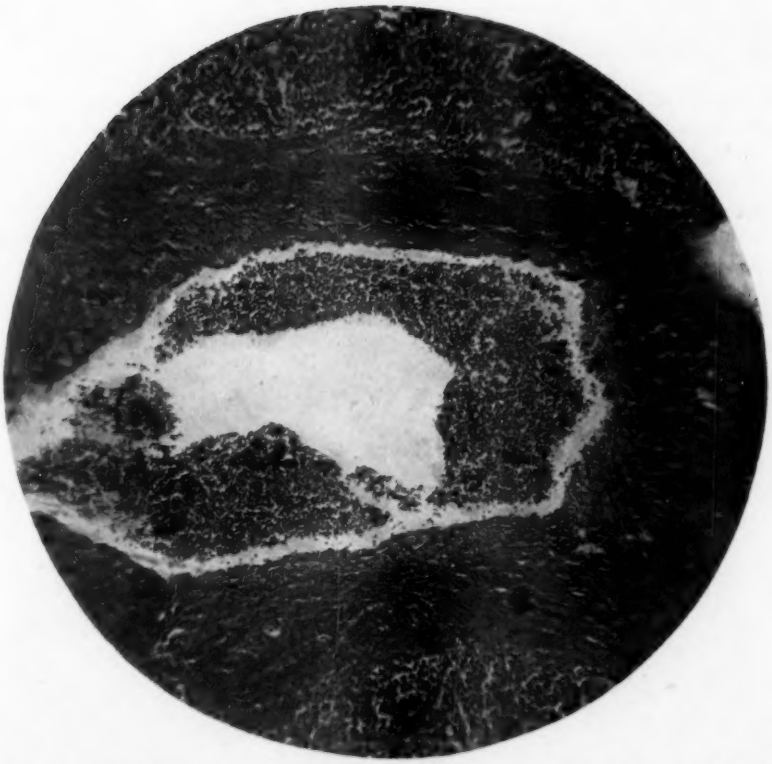


Fig. 3.—Dilated vein in the spleen with thickened wall. Hematoxylin-eosin; reduced from a magnification of × 100.

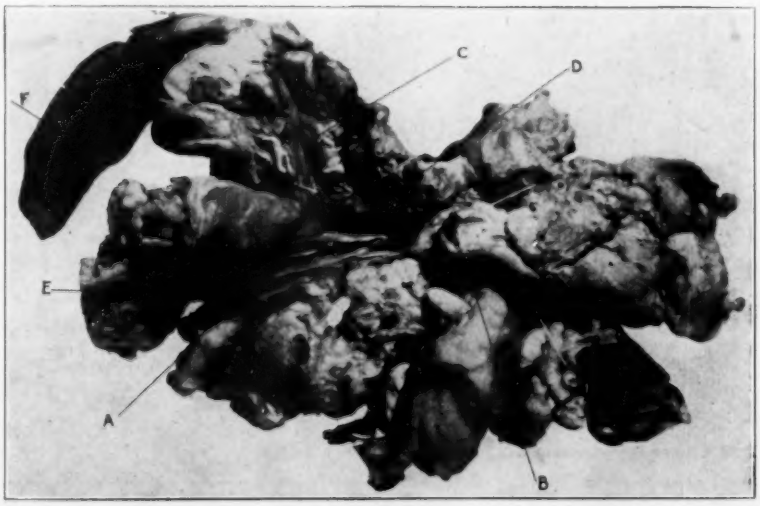


Fig. 4.—*A* indicates the dilated splenic vein; *B*, the dilated mesenteric vein; *C*, the narrowed portal vein; *D*, the dilated portal vein; *E*, the head of the pancreas, and *F*, the gallbladder.

after admission, the spinal fluid was clear, colorless and under normal pressure. The Pandy test yielded a negative reaction. Fehling's test showed a reduction. There were 4 cells per cubic millimeter (lymphocytes); a smear was negative and a culture sterile.

Clinical Course.—The temperature was 103 F. on admission, the pulse rate 140 and respirations 70. An immediate transfusion of 250 cc. of unmodified blood was given, with a resulting good color, but there was no improvement in the general condition. The prostration continued, and sordes were present about the buccal cavity. The temperature ranged between 103 and 105 F. for two

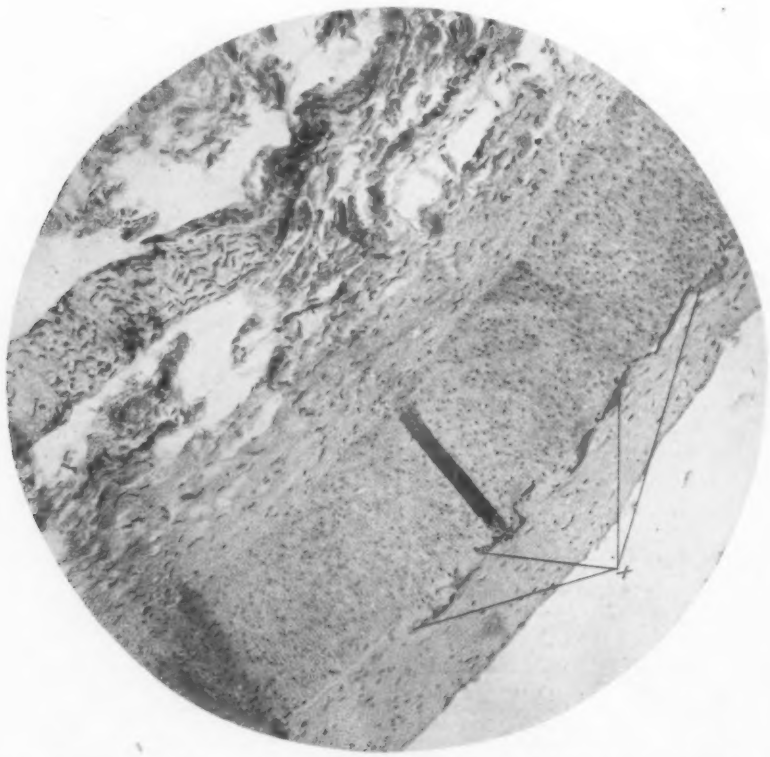


Fig. 5.—Wall of the portal vein, with thickening and fibrous changes in the muscularis, fraying and thickening and discontinuity of the elastica; thickening by fibrous deposit of intima. *X* indicates the elastica. Hematoxylin-eosin; reduced from a magnification of × 100.

days, declined to 101 F. for two days and then rose to 103.8 F. terminally. There was no reaction to active stimulation, nor did the abdominal distention diminish in spite of vigorous treatment. No fluid was present in the abdomen. The condition at this time was thought to be thrombosis of the splenic vein, in view of the large spleen, the abdominal distention, the anemia, the normal blood picture, the absence of purpuric spots and the repeated gastric hemorrhages. The patient was treated for dehydration by means of clyses. On one occasion, three days after admission, a tetanic-like convulsion occurred, for which calcium

chloride was given orally. The ear drums were still bulging, but the opinion of the otologist was that the general condition rendered it inadvisable to incise them. The examination of the fundus disclosed nothing abnormal except a pallor of the papillae. The temperature and the rate of respiration began to rise on the day of death, the temperature reaching 103 F. and the respirations 100 per minute. Free fluid was made out in the abdomen, shifting dulness being present; generalized rigidity developed. The chest still showed a few scattered râles. Death occurred six days after the second admission.

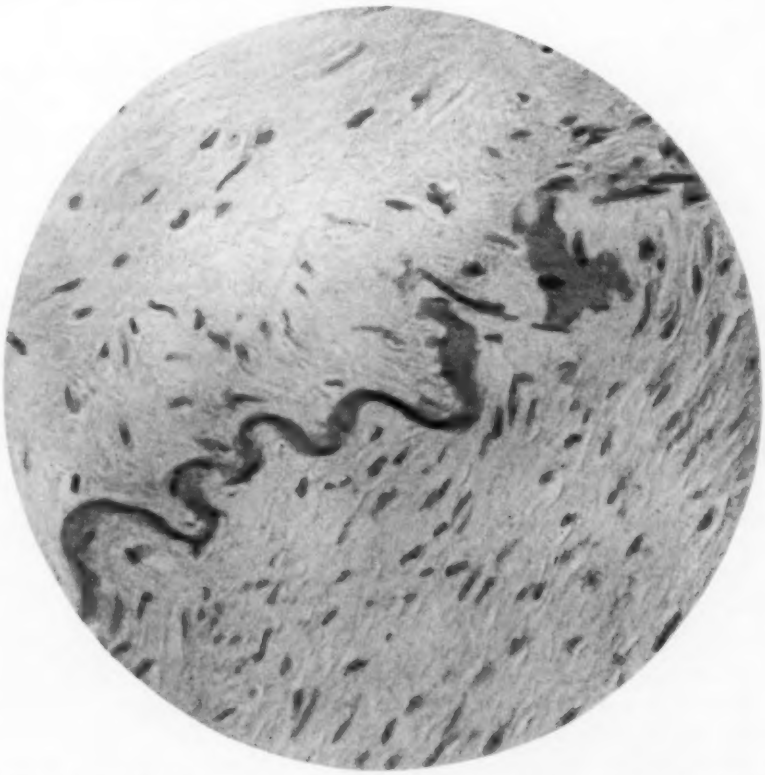


Fig. 6.—Detail of the intima and elastica. Note the broadening and defects. The upper layer is the intima. Hematoxylin-eosin; reduced from a magnification of × 400.

Autopsy.—The meninges appeared to be normal. The left middle ear and the ethmoid sinuses contained pus. The abdomen was markedly distended, and the peritoneal cavity showed a purulent plastic exudate. There were fresh adhesions between the coils of intestine. There was a marked thickening of the peritoneum and the omentum about the spleen and about the pancreas and duodenum. Microscopically, the lungs showed marked edema and congestion, with a compensatory emphysema and rupture of air vesicles. The heart was normal. The liver weighed 450 Gm. The edge of the right lobe was covered

with a thick, purulent exudate which could be easily scraped off. The surface otherwise was smooth and yellow, with mottled dark red areas. It was firm and cut with normal resistance. The parenchyma appeared pale and yellow. The connective tissue markings were prominent. Microscopically, the liver showed marked fatty degeneration and infiltration. Only a few of the liver cells were preserved. They were situated in the center of the lobules (fig. 1).

The spleen weighed 124 Gm. It was covered with a thick plastic exudate which could not be removed entirely. It was slate gray and firm. It cut with increased resistance, and the connective tissue markings appeared prominent. Microscopically, it showed marked thickening of the capsule with a definite increase in fibrous tissue. The trabeculae throughout the spleen were markedly enlarged and fibrotic. There was marked congestion, with areas of hemorrhage and compression of the follicles. The media of the vessel walls showed fibrosis and edema. The sinus walls were thick and fibrotic (fig. 2). The veins were greatly dilated (fig. 3). The esophagus appeared normal in size. On section the upper one third of the mucous membrane appeared pale and normal. The lower two thirds showed prominent and varicose esophageal veins with a congested mucous membrane. Microscopic examination showed destruction of the mucous membrane at one point. There was marked dilatation and congestion of the blood vessels with formation of thrombi. The splenic vein was normal in size, not thrombosed, and coursed through the body of the pancreas to join the superior mesenteric vein, thus forming the portal vein. The latter appeared normal in size at the junction of the splenic and superior mesenteric veins, but as it pursued its course toward the liver, its lumen became much smaller than normal and narrowed to about 2 mm. in diameter (fig. 4). Microscopic examination of the portal vein showed marked thickening of the intima and media, with fibrous connective tissue proliferation in these coats. The elastic fibers were irregular in outline and had disappeared in some places (figs. 5 and 6). Examination of the bone-marrow showed the megakaryocytes normal in number, erythrogenesis normal and leukogenesis increased, especially that of the eosinophilic myelocytes.

The anatomic diagnosis was: purulent peritonitis; pulmonary edema, congestion and emphysema; fibrosis of the spleen; congestion and cloudy swelling of the kidney; necrosis in the suprarenal glands; fatty degeneration of the liver; congestion and varicosity of veins in the esophagus with scarring and portal phlebosclerosis, with complete occlusion of the portal vein. The clinical impression was that of a case of atresia of the portal vein due to phlebosclerosis associated with esophageal varices, splenomegaly, terminal purulent plastic peritonitis, bronchopneumonia, left otitis media purulenta and ethmoid sinusitis.

COMMENT

Gross Pathology.—Most of the cases reported in the literature showed at autopsy or during the operation for splenectomy the lesions found in this case. Thus, Bardach's case [2] revealed at autopsy varicose veins in the lower half of the esophagus and in the fundus of the stomach. The duodenum, pancreas and structures at the hilum of the liver were matted together by a fibrous structure through which the portal vein passed, causing a constriction of the latter with a

2. Bardach, Martha: Arch. f. Kinderh. **71**:270, 1922.

secondary thrombosis of the splenic vein, splenomegaly and all the symptoms of portal occlusion.

Wahlgren's four cases [3] also showed either obstruction from without or narrowing from within, and adhesions were found between the spleen and the stomach. The lumen of the portal vein in one of the patients was only 3 mm. In our case it was 2 mm.

In one case Galloway [4] found the obstructive lesion mainly at the point of junction of the splenic and mesenteric veins. In the foregoing case the obstruction was found in the trunk of the portal vein. The gross pathologic lesions are not easily demonstrable during an operation for splenectomy, and removal of the spleen usually fails to indicate the site of the causative lesion. Wahlgren [3] did not find it in the intrahepatic branches but always in the peripheral branches of the portal vein, where the stenosis is followed by portal stasis and thrombosis.

Microscopic Pathology.—In the foregoing case the portal branches in the liver showed no changes. The lesion was found in the main portal trunk, as described, and consisted microscopically of phlebosclerosis.[5] Adami [6] stated that this type of lesion is not associated with any inflammatory condition, but that it is a strain fibrosis not due to irritation. It is more of a physiologic than of a pathologic nature. The walls are thicker, and the connective tissue is hypertrophied in all the layers. In the media there are tears and degeneration of the elastic fibers, and one may find deposits of lime. In the intima the connective tissue is poor in nuclei. Benda,[7] on the other hand, considered it as a compensatory thickening that develops on the basis of a variety of degenerative and inflammatory processes. Simmonds [8] did not find any evidences of inflammation. The portal vein resembles an artery in many respects. It has no valves and has a contractile capacity with muscle fibers similar to an artery. Portal sclerosis was therefore considered analogous to atherosclerosis. In the former, however, there are rarely any deposits of lipoid. It was thought, therefore, that two different causes operate, the chemical in atherosclerosis and the mechanical in phlebosclerosis. To prove this,

3. Wahlgren: Acta paediat. 1927, supplement, vol. 6.

4. Galloway, J.: Brit. J. Child. Dis. **13**:64, 1916.

5. Bruning and Schwalbe: Handbuch der algemeinen Pathologie und der pathologischen Anatomie des Kindesalter, Munich, J. F. Bergmann, 1924, vol. 2, p. 1225.

6. Adami, in Allbutt and Rollston: System of Medicine, 1912, vol. 1, p. 799.

7. Benda, in Hencke and Lubarsch: Handbuch der speziellen pathologische Anatomie und Histologie, Berlin, Julius Springer, 1924, vol. 2, p. 829.

8. Simmonds, cited by Benda, in Aschoff, Ludwig: Pathologische Anatomie, Leipzig, Johann Ambrosius Barth, 1921, vol. 2, p. 91.

Wohlwill and Holm [9] produced increased portal pressure in experimental animals by anastomosing the portal vein to the inferior vena cava and ligating the latter above the site of anastomosis, thus directing all the venous blood from the lower extremities into the portal vein. They were able to demonstrate localized thickenings. When cholesterol, however, was injected into the veins of the lower extremities in these animals, the characteristic lesions described were obtained. This proves that both factors operate. The liver in our case showed congestion and fatty degeneration, probably due to the terminal infection. Eppinger [10] did not find any other lesions in this organ, for the hepatic artery, which supplies it, is not involved in the process. The diminished amount of portal blood reaching the liver may influence the secretion of bile. That effect, however, has not been demonstrated, and cases lasting as long as fourteen years have not shown any changes in the liver.

The marked anemia is considered by most observers to be due to the profuse hemorrhages. Bardach [2] thought that the presence of pyknotic nuclei in the splenic pulp was evidence also of a marked destruction of erythrocytes in the spleen. Kleinschmidt [11] also ascribed it to an exhaustion of the bone-marrow, but Opitz [12] cited against this explanation the fact that after splenectomy the number of red blood cells is increased. In our case the bone-marrow was fairly normal, which was in agreement with Eppinger's observations. The leukopenia and thrombocytopenia are probably caused by the congestive conditions in the spleen, for there is an increase in both elements after splenectomy. It is still debatable whether these conditions are due to a destruction or to a diminished production of these elements.

The spleen shows all the well known changes of marked congestion: hypertrophy and fibrosis of the capsule and trabeculae and thickening of the vessel walls with compression of the malpighian bodies. This congestion was due to the narrowed portal vein and subsequent interference with the normal flow of blood from the spleen. The blood in these cases reached the general circulation by way of a newly established collateral circulation. This may be one or more of the well known routes, depending on the localization of the lesion in the portal or splenic veins. Thus, if the obstruction is in the portal vein, the collateral circulation may involve the epigastric and internal mammary veins; the superficial abdominal veins then dilate and a caput medusae is formed. The gastric and esophageal veins may form

9. Wohlwill and Holm: Verhandl. d. deutsch. path. Gesellsch. **22**:235, 1927.

10. Eppinger, Hans: Die Hepato-Lienalen Erkrankungen, Berlin, Julius Springer, 1920, p. 399.

11. Kleinschmidt: Monatschr. f. Kinderh. **13**:505, 1914.

12. Opitz, Hans: Jahrb. f. Kinderh. **107**:211, 1924.

a collateral circulation, owing to back pressure in the spleen. These veins become engorged and thrombotic, and form varices which rupture on further increased pressure, resulting in hemorrhage. The low platelet count, interfering with clot formation, also favors hemorrhage. This was the modus operandi in the foregoing case. There was another collateral circulation at play in our case, namely, the inferior mesenteric vein with the veins of the hemorrhoidal plexus, as evidenced by the occasional rectal bleeding.

Ascites was not present in this case during the patient's first stay in the hospital, but was noted at the end of the second stay. This condition is not always present in these cases, and when present is due to the marked anemia, the poor general circulation and stasis. The terminal peritoneal infection was responsible for this condition in our case. It is true, though, that ascites is more frequently associated with portal than with splenic thrombosis. One of Opitz' cases [12] of splenic thrombosis showed no ascites, while another case showed ascites and dilated abdominal veins and was probably due to a lesion of the portal vein.

There is usually no elevation of temperature, unless an intercurrent or terminal infection occurs, as in this case. Galloway [4] reported a case in which the patient had a high temperature which he thought was due to the thrombosis spreading into portions of the portal circulation away from the portal vein.

Etiology.—Any intra-abdominal infection leading to a mesenteric thrombosis may cause portal thrombosis by an embolus. Thus, postoperative intra-abdominal infections and typhoid fever have been known to be causative factors. These are out of consideration in the foregoing case, as are also umbilical granulomas, infections and furunculosis during the neonatal period, as emphasized by Kleinschmidt [11] and Wahlgren [3] in their cases, in which the infection traveled by way of the umbilical vein to the portal vein where the lesion was found. As far as could be ascertained, the patient did not have any umbilical infections or skin lesions during the period following birth. Klemperer [13] reported a case of portal thrombosis due to alteration in the composition of the blood; i. e., a thrombocythemia, in which multiple thrombi were also found in other vessels. The number of platelets in our case was never above normal, and the bone-marrow showed a normal number of megakaryocytes. Wahlgren [3] reported one case in a geophagist, in whom he found a thrombosis in the gastric veins due to injury of the gastric mucous membrane by the ingested material. The patient in the case described did not have this habit.

---

13. Klemperer: Cavernomatous Transformation of Portal Vein; Its Relation to Banti's Disease, Arch. Path. **6**:353 (Sept.) 1925.

Smith and Howard [14] stated that portal obstruction may come about not only from sclerosis following a phlebitis but also from adhesions about the vein after a plastic peritonitis, from pressure from without, or from abnormalities of the vessel itself. In Klemperer's case [13] the portal obstruction became cavernous, and vascular tissue formation was located in the hepatoduodenal ligament. Hecht showed anomalies in the positions of the vessels at the hilum of the liver that might be congenital or due to a contracting exudate. Gruber [15] showed a congenital portal stenosis in his case. The latter condition is the one which existed in our case, although there were also many thick fibrous bands, probably the remains of some intra-abdominal pathologic condition during intrauterine life.

Clinical Course.—This condition usually continues, with remissions, for a long time without any enlargement of the liver or ascites, but presents splenomegaly, anemia with leukopenia and recurrent hemorrhages. During long remissions the anemia may disappear and the patient may appear normal, but the splenomegaly continues. The splenic tumor usually becomes smaller a few days after a hemorrhage and then begins to increase in size again. This causal interrelation of the splenomegaly and hemorrhages was noted by Opitz [12] in his two cases and was emphasized as of great diagnostic importance.

The hemorrhages may be preceded or accompanied by abdominal pains, as in Bardach's case.[2] Early in the course of the disease, the hemorrhage may be slight and may occur in the form of an occasional epistaxis, while later in the disease the hemorrhage may become severe, in the form of hematemesis, and if not controlled rapidly, may lead to a fatality.

Differential Diagnosis.—The cardinal points on which the diagnosis of portal phlebosclerosis should be made are the splenomegaly, the anemia, the signs of collateral circulation, such as dilated superficial abdominal veins, and the copious hemorrhages which are usually followed by a diminution in the size of the spleen. The leukopenia and the occasional thrombocytopenia are also to be looked for, but are not always present. The diagnosis of Banti's disease, as stated in the beginning of this communication, is frequently made in this condition because of the similar blood picture. Banti's disease, however, is rare in infancy and childhood, and usually a large liver, ascites and urobilinuria are associated with it; the splenomegaly is progressive and is not related to the hemorrhages. Gastric ulcer is also rare in infants and children, and pain is usually present. Pernicious anemia shows a

14. Smith and Howard: Early Occurrence of Gastric Hemorrhage in Children with Splenomegaly, Am. J. Dis. Child. **34**:585 (Oct.) 1927.

15. Gruber, G. B.: Deutsches Arch. f. klin. Med. **122**:319, 1917.

definite blood picture and is also infrequent at this age. In leukemia the blood picture is characteristic. There are usually enlarged lymph nodes, and the platelets and leukocytes are not reduced. The condition does not last long. The anemias due to chronic intoxications or metabolic disturbances, such as rickets, alimentary anemia and von Jaksch's disease, can be ruled out by the observance of normal bone and normal calcium and phosphorus levels in the blood. Examination of the stools for ova and of the blood smear for eosinophilia will exclude anemia due to infestation by worms. The absence of glandular enlargements and the presence of hematemesis will rule out Hodgkin's disease, although leukopenia may be present in the abdominal form of Hodgkin's disease. In Henoch's purpura, the presence of arthritic and skin manifestations, the absence of leukopenia and the usual occurrence of hemorrhages from the large intestines make an error in diagnosis improbable. It is true, though, that the chronic course, the periods of well-being and the few purpuric areas over the extremities make one suspicious of purpura, but the spleen in the latter disease is not so enlarged. Werlhof's disease can be ruled out by the absence of a marked thrombocytopenia, by the skin manifestations and by the fact that splenomegaly is not a sign of this condition. Gaucher's disease or Niemann-Pick splenomegaly and the primary (isolated) splenic tuberculosis are not associated with hematemesis; in the latter disease, there is usually hyperglobulia.

Prognosis.—In portal thrombosis a spontaneous cure may occur by canalization of the thrombus or by the relief of the obstruction. This can scarcely occur in a congenital narrowing and sclerosis of the vein, and the outlook is therefore unfavorable unless heroic measures are undertaken to check the increased back pressure.

Treatment.—Usually the patient presents himself after a severe hemorrhage, when he has marked anemia. A transfusion to relieve this condition temporarily is the proper procedure. Some observers advise frequent small or large transfusions and a diet rich in iron, while most of the recent observers advise splenectomy. Kleinschmidt,[11] in 1914, was opposed to splenectomy; he thought that it would not benefit the condition, as the lesion was not in the spleen. Smith and Howard,[14] on the other hand, thought splenectomy beneficial, for there would be a diminution in the amount of blood going through the portal vein and there would therefore be less engorgement. They reviewed twenty-six cases in the literature (including five of their own), in eleven of which splenectomy was performed. Of these eleven patients, ten are alive and well and have not had recurrent hemorrhages; the eleventh died after operation. Of the fifteen patients on whom a splenectomy was not performed, nine died, several suddenly and others a few years later, and six are still alive. This speaks well for splenec-

tomy. We advised this procedure in our case after the first sojourn in the hospital, but the parents did not consent. During his second stay the patient was too ill to have the operation performed. Even after splenectomy we are not certain that hemorrhage may not recur.

CONCLUSION

A case of portal phlebosclerosis in a boy, aged 2 years, due to a congenital stenosis, is described. Observations at autopsy and microscopic data are given, together with comments on the symptomatology, etiology, pathology, differential diagnosis and treatment of the condition.

CONGENITAL ESSENTIAL THROMBOCYTOPENIA *

HARRY M. GREENWALD, M.D.
AND
IRVING SHERMAN, M.D.
BROOKLYN

Congenital essential thrombocytopenia or congenital morbus maculosus werlhofii is a rare condition. Rushmore reviewed the literature up to 1925 and collected six cases of congenital thrombocytopenia. In the cases collected by Rushmore [1] and in that reported by Liebling,[2] the mothers also manifested symptoms of thrombocytopenia during the last few months of pregnancy. Waltner [3] observed a case in a new-born premature infant whose mother had had signs and symptoms of thrombocytopenic purpura since she was 15 years of age. We observed a case of essential thrombocytopenia in a new-born infant who died fourteen days after birth. Observations at necropsy permitted us to draw conclusions as to the origin of the pathologic changes in the blood. These conclusions will be discussed subsequently.

REPORT OF CASE

History.—J. K., a boy, was born in the United Israel Zion Hospital on Dec. 13, 1928. Delivery was normal; the infant cried lustily at birth and presented no evidence of bleeding. On the sixth day after birth a rash was observed about the infant's neck, back and shoulders. On the seventh day he was circumcised; no unusual amount of bleeding was noted. He was discharged from the hospital on the tenth day after birth. Four days later, the attending physician asked one of us to see the infant. At this time, December 27, physical examination revealed the following condition: The infant looked acutely ill, the respirations were rapid and stertorous and cyanosis of the lips and finger-nails was present to a marked degree. The rectal temperature was 98.2 F. There was a purpuric eruption on the face, shoulders, arms, trunk and extremities; the eruption was bright red and varied in size from that of a pinhead to that of a pea. There were also hemorrhages on the buccal mucous membrane. Blood was oozing from the circumcised area, which had not entirely healed, and from the rectum. Examination of several stools revealed a fairly large amount of blood. The fontanel was somewhat depressed. The heart sounds were not audible because of the loudness and harshness of the breath sounds. The abdomen was somewhat distended. The liver was palpable about a fingerbreadth below the ribs, but the edge of the spleen was not felt below the costal margin. A tentative diagnosis of thrombocytopenic purpura was made,

* Submitted for publication, May 4, 1929.

* From the Departments of Pediatrics and Pathology, United Israel Zion Hospital.

1. Rushmore, S.: Am. J. Obst. & Gynec. **10**:553 (Oct.) 1925.
2. Liebling, P.: Am. J. Obst. & Gynec. **11**:847, 1926.
3. Waltner, K.: Jahrb. f. Kinderh. **106**:307, 1924.

and immediate hospitalization was advised. The infant was readmitted to the hospital one-half hour later. Physical examination on admission revealed essentially the same observations, except that no radial pulse could be obtained. The longitudinal sinus was punctured and about 5 cc. of blood removed for examination. The child died about one and a half hours after admission to the hospital. Examination of the blood revealed the following: red blood cells, 6,100,000; hemoglobin content (Sahli), 122 per cent; white blood cells, 12,000; 57 per cent polymorphonuclear leukocytes; 41 per cent lymphocytes; 1 per cent myelocytes, and 1 per cent transitionals. There were 15 nucleated red blood cells. The coagulation time, by the capillary tube method, was thirty-five minutes and the bleeding time five minutes. There was no retraction of the clot at the end of twenty-four hours. There were

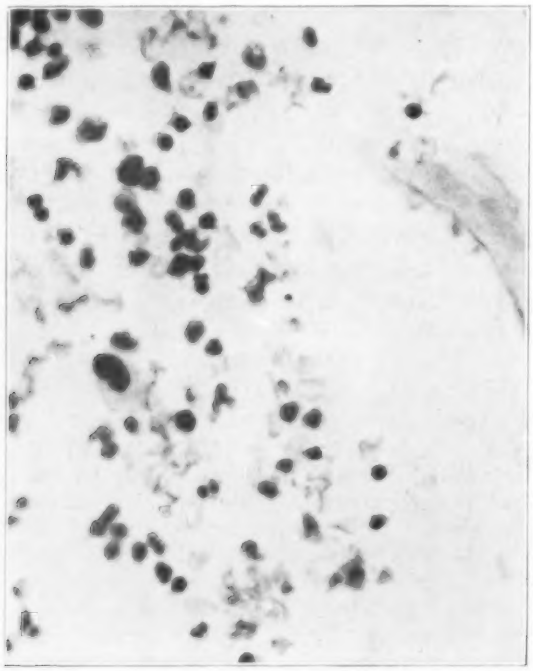


Fig. 1.—Bone-marrow. Wright stain. Leitz Apo. 4 mm. Periplane eyepiece 6. There is one small megakaryocyte with a bean-shaped nucleus.

30,000 platelets present. The Wassermann reaction was negative. The capillary resistance test was positive; in the fragility test beginning hemolysis was revealed at 40; it was incomplete at 30, showing increased resistance.

Necropsy.—A necropsy was performed by Dr. Goldzieher, who gave the following report: The body was that of a well developed male infant with no gross abnormalities. There were small petechial hemorrhages covering the face, arms, upper part of the thorax and lower extremities. The patient was markedly blue, particularly about the nose, lips, face and hands. When the body was opened, about a pint of cloudy yellow fluid was found in the peritoneal cavity. Both the parietal peritoneum and the serosa of the intestines were shiny and pale. In view of the fact that there was no evidence of inflammation of the peritoneum, a bacteriologic study of the ascitic fluid was not made.

The brain was soft and revealed no hemorrhages in any part.

The lungs were grayish-red and contained air throughout. On the surface of the right lung, a number of small petechial hemorrhages were seen. On section, a reddish fluid was exuded.

The heart was large, particularly in its transverse diameter. The left ventricle was a mere appendage to the right side of the heart; its wall was thin and its lumen only a narrow rent. The left auricle communicated freely with the right auricle. As a matter of fact, there was only a small band, partly fibrous and partly muscular, separating the two auricles; below this band there was also a communication between the two ventricles.

The musculature of the right ventricle was considerably thickened. The pulmonary artery was large and was three times the size of the aorta. The ductus was wide and patent, and led into the arch of the aorta, which appeared to be the

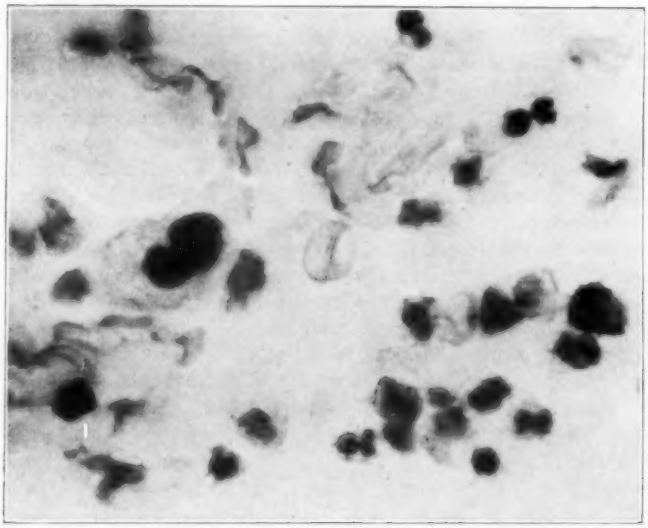


Fig. 2.—The same slide as in figure 1. Leitz Apo. 2 mm. Periplane eyepiece 6. Cytoplasm of the cell; the same megakaryocyte shows no granulation.

continuation of the wide pulmonary artery. The valves of the heart were thin and smooth.

The spleen was medium-sized, firm and dark red.

The liver was somewhat enlarged and firm; its section had the appearance of nutmeg.

The mucosa of the stomach and intestines was dark red, swollen and covered with mucus.

The kidneys were of medium size, firm and dark reddish gray; their capsules stripped off readily.

The suprarenal glands were flat and rather small; their structure was well maintained. The bone-marrow was dark reddish gray.

Diagnosis.—The diagnosis was: congenital anomaly of the heart, communication of both ventricles and auricles, open ductus botalli, chronic passive congestion of all the viscera, ascites and thrombocytopenia.

Histologic Description.—The spleen showed numerous small follicles but no secondary follicles; they were sharply demarcated from the red pulp which was congested. The sinuses were also engorged; the endothelium of the sinuses was flat and the pulp was not very cellular. There was no evidence of increased phagocytosis either by the reticulum cells or by the sinus endothelium.

Congestion of the liver was conspicuous. There were irregular areas which usually occupied the center of the lobules or were scattered in no apparent plan. Liver tissue was absent in the areas which were occupied only by the distended capillaries. The latter contained a number of leukocytes. Swelling of the Kupffer cells was noteworthy; they were mostly loaded with brownish pigment and were distinctly different from those outside the congested territories. The latter were rather numerous but unusually large, and did not reveal evidence of enhanced phagocytic activity. Neither Glisson's capsule nor the peritoneal capsule of the spleen showed any evidence of inflammation.

The thymus gland consisted mostly of fat tissue which enclosed a few small lobules of thymic parenchyma. These glandular islands were of unsual structure in that they did not contain a single Hassall's corpuscle. Differentiation between cortical and medullary portions was absent. Only occasionally could small areas with more densely packed, small lymphocyte-like cells be distinguished. Most of the tissue consisted of cells with pale oval nuclei, pertaining apparently to the medulla. Scattered in between these cells were leukocytes, eosinophilic myelocytes and lymphocytes. The tissue was well vascularized and the vessels were engorged.

The bone-marrow was fairly cellular with areas of excessive cellularity in which the spaces between the bone trabeculae were densely jammed. The large sinuses were engorged. There were also many red blood cells in the meshes of the marrow. The majority of the cells consisted of myelocytes among which the eosinophils were unusually numerous. Mast cells were also fairly common. Among the reticulum cells there were some which stood out because of their unusually large size; however, they did not show any evidence of excessive phagocytosis. Other cells included mature polymorphonuclear leukocytes, lymphocytes, normoblasts and cells more embryonal in type, which might be classed as myeloblasts or hemocytoblasts.

Megakaryocytes were extremely scant. They were comparatively small. This applied particularly to the nuclei which were conspicuously hyperchromatic. Some of these cells had an oval or bean-shaped nucleus which was different from that of the ordinary megakaryocyte. The cytoplasm of some of the cells was scant, but in others fairly well developed; it stained plain blue and was devoid of the granules usually characteristic of the mature megakaryocyte.

COMMENT

Thrombocytopenic purpura can be divided into two main groups: symptomatic and essential thrombocytopenia. The first group includes all those cases of thrombocytopenic purpura in which the insufficiency of platelets is just a symptom of a severe general condition. Symptomatic thrombocytopenic purpura may be subdivided into four groups: (*a*) thrombocytopenia following dyscrasia of the blood (leukemia, pernicious anemia, aplastic anemia and agranulocytosis); (*b*) thrombocytopenia following acute infections (general sepsis, subacute bacterial endocarditis, typhus fever, etc.); (*c*) thrombocytopenia following poisoning with organic chemicals, such as benzol, benzine or arsenic compounds, and

(*d*) thrombocytopenia subsequent to destruction of bone marrow by granulation tissue (Hodgkin's disease, neoplasm or hyperplastic proliferation of the reticulum cells (Gaucher's disease). Other factors in symptomatic thrombocytopenic purpura possibly include anaphylactic phenomena and the effect of lack of vitamins.

The second group, that of essential thrombocytopenic purpura, is characterized by the absence of any other disease or known impairing factor which can be held responsible for the lack of platelets.

There are two theories as to the mechanism through which the reduction of the platelets occurs. The first is that of Kaznelson,[4] who stated that it was due to a primary hyperfunction of the spleen with a destruction of the platelets. He believed that the bone-marrow was intact and performed its normal hemopoietic functions. For this reason he termed the disease thrombolytic purpura. Frank,[5] on the other hand, said that there was a defect in the production of the platelets which he attributed to the effect of some noxious agent on the megakaryocytes.

Experimental proof and observations at necropsy were employed by both authors in support of their respective theories. The number of examinations of bone-marrow, either by autopsy or by sternal puncture, has been limited. Foerster,[6] Sternberg [7] and Kaznelson [8] found a large number of megakaryocytes; in fact, Kaznelson found the megakaryocytes with complete functions, as evidenced by an abundant cytoplasm and a large number of growths, in a patient who had died after splenectomy. On the other hand, Frank [9] and Stern and Hartman [10] found definite morphologic changes in the megakaryocytes on sternal puncture, particularly an absence of the azure granules. Recently, Lautier,[11] Oberling and Woringer found an absence of megakaryocytes in a boy, aged 5 years, who died one week after splenectomy; however, myeloblasts, myelocytes and erythroblasts were present in large numbers.

The constitutional factor in essential thrombocytopenia is emphasized in the literature of the last few years. In fact, Glanzmann [12] spoke of a hereditary hemorrhagic thrombo-asthenia, which is, perhaps, as common a constitutional anomaly as exudative diathesis; it is his belief that morbus maculosus werlhofii is an unusual manifest type of the thrombo-

4. Kaznelson: Ztschr. f. klin. Med. **87**:133, 1919.
5. Frank: Die hamorrhagischen Diathesen in Schittenhelms Krankheit des Blutes und Blutbildende Organe, Berlin, Julius Springer, 1925.
6. Foerster, A.: Ztschr. f. klin. Med. **92**:170, 1921.
7. Sternberg, F.: Wien. Arch. f. inn. Med. **3**:433, 1922.
8. Kaznelson: Wien. Arch. f. inn. Med. **7**:87, 1924.
9. Frank, quoted by Kaznelson (footnote 4).
10. Stern and Hartman: Ztschr. f. klin. Med. **104**:490, 1926.
11. Lautier, Oberling and Woringer: Rev. franç. de pédiat. **3**:340, 1927.
12. Glanzmann: Jahrb. f. Kinderh. **88**:1, 1919.

asthenic diathesis. Glanzmann examined several members of a family with this constitutional anomaly and found morphologic changes in the platelets in the presence of a normal count, with absent or incomplete retraction of the blood clot. Leschke,[13] too, considered the cause of this disease a constitutional insufficiency of thrombocytogenesis, and he stated that he saw two cases in which new-born infants showed evidences of this disease immediately after birth. In one case the mother also suffered from thrombocytopenia. In the cases reported by Liebling,[2] Waltner [3] and Rushmore,[1] the mothers also suffered from the disease. Unfortunately, in our case we were unable to obtain a specimen of blood from the mother for examination.

We consider the present case one of essential thrombocytopenic purpura, as none of the factors could be demonstrated that are known to be instrumental in producing symptomatic thrombocytopenic purpura. Our case is noteworthy for the definite changes in the bone-marrow, and for the coincidence with congenital anomalies of the heart and thymus gland.

The great scarcity of megakaryocytes, their poor development and the absence of their specific granulations is well in keeping with the observations of other writers. This would substantiate the conception of Frank [9] that thrombocytopenia depends on the lack of megakaryocytes or on the inadequate function of these cells, as visualized by the absence of their granules.

The other conception of thrombocytopenic purpura, according to which excessive destruction of platelets is responsible for their diminution, is not corroborated in our case by histologic evidence. Neither the cells of the splenoreticulum or the sinus endothelium of the spleen nor the Kupffer cells showed any signs of excessive phagocytic activity. Phagocytosis by Kupffer cells was also observed only in those areas in which destruction of red cells and of the parenchyma of the liver occurred, owing to passive congestion. The latter is an obvious consequence of the cardiac lesion.

Although the first signs of the thrombocytopenic syndrome were witnessed six days after birth, it is our opinion that the scarcity of megakaryocytes and their visible morphologic impairment, which we hold responsible for the thrombocytopenia, were already present at birth. In the period between birth and the onset of symptoms no intercurrent disease, and notably no infection, occurred. At no time during the first ten days of life and during the period following readmission was there any elevation of temperature. Careful examination of the umbilicus showed no evidence of infection anywhere; the abdomen, although somewhat distended, was not tender. Histologic exam-

13. Leschke: Essentielle Thrombopenie, Ergebn. d. ges. Med. **9**:344, 1926.

ination of the spleen ruled out the possibility of an acute infectious process, since the polymorphonuclear leukocytes in the splenic pulp were scarce and changes in the splenic reticulum were lacking that are characteristic of infectious conditions.[14] In addition, no bacteria were found in the special stained sections of the marrow of the bone and spleen.

It seems logical to assume, therefore, that this is a case of congenital thrombocytopenia based on an abnormal development of bone marrow. This coincides with the other anomalies of development, namely, those of the heart and the thymus gland.

We do not desire to enter into a discussion of the relationship between the manifest errors of development (heart and thymus gland) and the lesion of the bone-marrow. This coincidence, however, seems to support the assumption that the defective formation of megakaryocytes was congenital.

The high red blood cell count and normal bleeding time are not the usual observations in a case of thrombocytopenia. The high red blood cell count can easily be explained by the age of the infant and by the effect of the congenital cardiac lesion. The short bleeding time may have been due to venous stasis and the failing peripheral circulation at the time that the test was made, although there are other cases of thrombocytopenic purpura reported in the literature in which the bleeding time was normal or but slightly increased (Liebling,[2] Rosenthal,[15] Roskam [16]).

CONCLUSIONS

1. A case of congenital thrombocytopenic purpura is reported.

2. The reduction in the number of the platelets is attributed to an insufficiency of megakaryocytes of the bone-marrow.

3. The coincidence of anomalies of the heart and of the thymus gland with defective formation of the megakaryocytes supports the conception of the congenital nature of the thrombocytopenic purpura.

---

14. Goldzieher and Lurie: Experimental Studies on the Reticulo-Endothelial Septum, Arch. Path. **7**: 646 (April) 1929.

15. Rosenthal, N.: J. Lab. & Clin. Med. **13**:303, 1928.

16. Roskam: Presse méd. **31**:472, 1923.

PNEUMONIC ANTHRAX IN A CHILD*

J. C. McKITTERICK, M.D.
AND
GEORGE J. PEARSON, M.D.
BURLINGTON, IOWA

History.—A girl, aged 2½ years, was admitted to the hospital on Dec. 10, 1928, with a condition that was diagnosed as pneumonia. There was no history of a previous influenza or even a bad cold, and the symptoms were said to have begun with a "choking spell" on December 8.

Examination.—Physical examination disclosed a fairly well nourished child, a temperature of 103 F., restlessness, coughing at intervals and a marked translucent edema about the eyes and extending to the waistline. The left eye was completely closed and the right eye was partially closed. On parting the lids, the pupils were found to be dilated, and there was some photophobia. Examination of the throat disclosed that it was swollen and red. Both ear drums were reddened and bulging; a paracentesis of both of the drums was done, a grayish-yellow purulent drainage resulting. The skin was mottled and partially cyanosed. Palpation revealed the fact that the edema was more of an emphysema, and on auscultation numerous fine crepitant râles were superimposed over tubular breathing. There was no marked dulness on percussion.

The abdomen was distended and was covered entirely by the emphysematous swelling, which included also the flanks and back. The urine was acid and contained a heavy cloud of albumin, many casts, some pus and red cells. The leukocyte count was 18,000. A roentgenogram of the chest was taken on the third day (fig. 1).

Treatment.—As it was thought that the child had bronchopneumonia with possibly pyopneumothorax, the usual supportive treatment was given. For the next three days the temperature fluctuated between 101 and 105 F., exhibiting a typically septic curve. On December 13, an exploratory puncture of the right side of the chest was made, and 5 cc. of blood-stained serum was removed. A gram stain of the smear of the fluid disclosed pneumococci. The next day, December 14, as the emphysematous condition was no better, the right side of the chest was punctured, and about 2 cc. of similar fluid was removed. No evidence of pneumothorax was discovered. Smears were made of this fluid, but before they were stained the specimen was discarded. These smears disclosed many gram-positive organisms morphologically similar to *B. anthracis.* We thought that this puncture was probably made into lung tissue, as we were never again able to secure a similar specimen even after repeated punctures. Park and Williams[1] said: "The detection of the anthrax bacillus is ordinarily not difficult, as this organism presents morphological, biological and pathogenic characteristics which distinguish it from all other bacteria. In the later stages of the disease, however, the bacilli may be absent or difficult to find and cultivation on artificial media and experimental

* Submitted for publication, July 11, 1929.

1. Park and Williams: Pathogenic Micro-Organisms, Philadelphia, Lea & Febiger, 1920, p. 481.

inoculation in animals are not always followed by positive results." Fluid taken at subsequent punctures showed no anthrax organisms, and had no effect when inoculated into guinea-pigs.

Considering that something should be done quickly for the patient, on December 14 an attempt was made to give horse serum intravenously, but owing to the subcutaneous emphysema it was not successful, and 20 cc. of plain horse serum

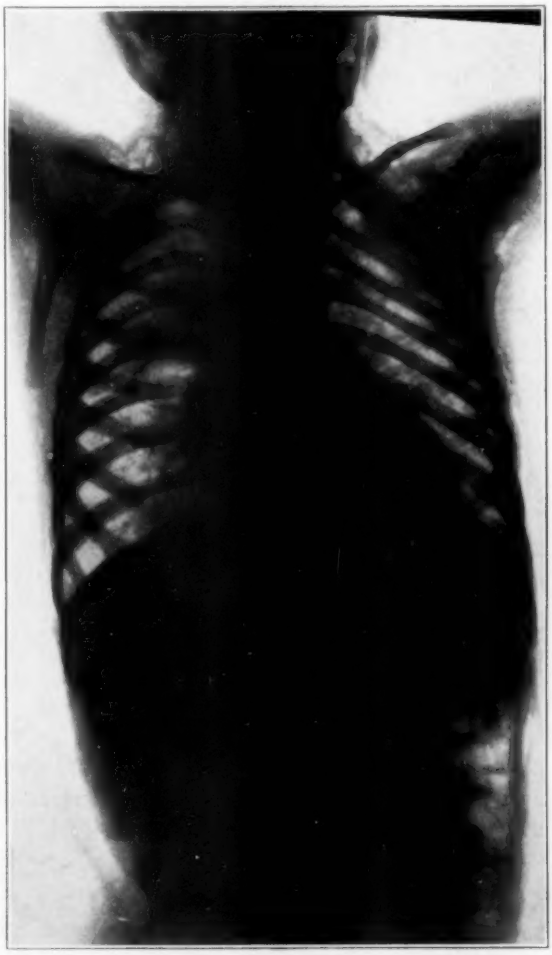


Fig. 1.—Roentgenogram of the chest taken on the third day after admission. There are emphysematous areas in the cellular tissues of the neck, face and axillae, and also of the abdominal walls. There is a diffuse pneumonic process of the right lung.

was injected intraperitoneally. On December 15, 16 and 18, respectively, 50 cc. of commercial anthrax serum was given intraperitoneally with the result that from the first injection the temperature curve rapidly declined, and by December 20 the temperature was normal and the emphysema practically gone. About this

time the child relished her food and seemed normal in every way, except that after the swelling left the region of the eye it was found that she could not see.

A report of the examination of the fundi is as follows: On December 22, examination of Marjorie Younge showed that she had widely dilated pupils which did not react to light. The media and coverings of the eye showed no pathologic process, no evidence of muscle paralysis and no disturbance of the muscular function. The fundi of both eyes were practically the same; that is, they showed a pale retina and almost white optic cup with no evidence of increased intra-ocular pressure. The veins could not be seen; the arteries showed only as minute threads. The conclusion was that there was occlusion of the optic arteries, probably by emboli. As the temperature was normal and the child was rapidly regaining strength, she was discharged from the hospital.

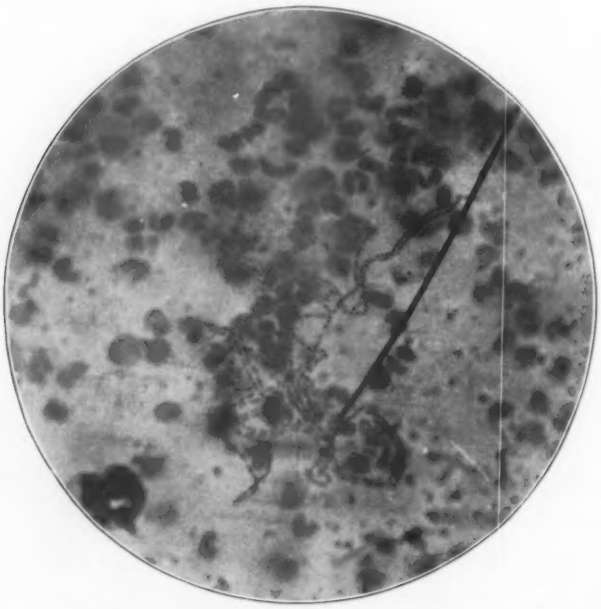


Fig. 2.—Photomicrograph of a typical group of the organisms found in the fluid from the chest. The size of the organisms can be estimated by comparison with the leukocytes. Gram's stain.

Course.—On Jan. 2, 1929, when she was seen again the same condition obtained in the eyegrounds. At this time, the parents said that on the farm on which they lived several horses had died within the past few years, their deaths being attributed to drinking water.

On January 24, the child was seen again. Improvement in strength, a good appetite and ability to walk with support were noted. The pupils were moderately dilated and reacted to light, and the child complained when strong light was thrown into the eyes. The retina was mottled, pink and white. There were no areas of pigmentation. The nerve head was grayish white, and the arteries were more easily seen than before, but still merely as threads. The pale spots in the retina were about the same color as the entire retina when first seen on Dec. 22, 1928.

On Feb. 8, 1929, she was again seen. Her appearance was that of a healthy child; she walked and talked, and gave evidence of light perception, but no vision. A specimen of the well water on the farm was examined bacteriologically, but nothing pathogenic was found.

Diagnosis.—In view, therefore, of these unusual physical observations of emphysema, cyanosis and great prostration, the discovery of typical anthrax organisms in fluid removed by lung puncture, the septic curve of the temperature, the rapid improvement on the administration of serum and the evidence of the lodgment of emboli in the ophthalmic arteries, the condition in this case was diagnosed as pneumonic anthrax (wool-sorters' disease).

213 Iowa State Bank Building.

CORRECTION

In the article entitled "Multiple Disseminated Lupus Vulgaris" by Vaughn C. Garner on page 1036 of the November issue, the first sentence under the heading, "Summary" should read:

1. "Multiple disseminated lupus vulgaris is a miliary form of tuberculosis of the skin in infancy and childhood."

Progress in Pediatrics

BILATERAL CONGENITAL FACIAL PARALYSIS

REVIEW OF THE LITERATURE AND A CLASSIFICATION *

BARNET E. BONAR, M.D.
SALT LAKE CITY
AND
RUSSELL W. OWENS, M.D.
MAGNA, UTAH

The term congenital facial paralysis is applied to a group of cases in which the paralysis is noticed at or shortly after birth. The definition is qualified in this way to include certain cases of undoubted congenital origin in which symptoms are not recognized at birth. The subject of congenital facial paralysis has had sufficient attention for one to assume a clearly defined picture for it, but, unfortunately on review of the literature one finds little clarity about the condition. This confusion has come about more from lack of understanding of the mechanism of development, together with the absence of a simple working outline, than from any other reason. In attempting to formulate such a classification, several factors must be taken into consideration. From a scientific standpoint, an anatomic classification might be more accurate, for instance, the division into supranuclear or nuclear lesions, lesions at the exit of the nerve from the pons, lesions in the aqueduct of Fallopius, and lesions peripheral to the exit of the nerve from the stylomastoid foramen, as is commonly given in treatises on facial paralysis in the adult. But this is not practical in small infants because of the difficulty in determining the status of hearing, taste and smell at this age. On the other hand, a classification based more or less on the cause makes the diagnosis more simple, localizes the lesion fairly accurately, and makes it possible to offer a definite prognosis with greater ease, a vitally important affair to the parents. From an etiologic consideration, there are two general groups into which these cases may be divided:

- Obstetric
  - Due to forceps trauma
  - Due to inherent pressure factors
    - Antepartum compression
    - Intrapartum compression
- Spontaneous or nonobstetric

* Submitted for publication, Sept. 27, 1929.

* From the Pediatric Department of the Holy Cross Hospital, Salt Lake City.

This classification, as far as the main headings are concerned, is similar to that of Falloux.[1] However, his subdivision of the nonobstetric variety includes one form of the paralysis which is of doubtful origin, that of agenesis of the mastoid region, suggested by Heller.[2] This division has been omitted, since in our opinion the cases so listed easily fit into other groups. This simplifies the classification greatly, and at the same time obviates any possible chance for argument about this group.

CONGENITAL FACIAL PARALYSIS OF OBSTETRIC ORIGIN

Mention of facial paralysis in the new-born usually leads one to think of the unilateral form caused by trauma to the extracranial portion of the seventh nerve during forceps deliveries. True it is that the great majority of paralyses are of this origin, but if it is realized that there are other forms, no great surprise need be manifest if such a case presents itself. Aside from the class due to pressure from the forceps blades, there is a group which follows noninstrumental deliveries.

Facial Paralysis Due to Forceps Trauma.—This is the unilateral form due to injury of the peripheral portion of the seventh nerve, either from direct pressure of the blades of obstetrical forceps on the nerve trunk after its emergence from the stylomastoid foramen, or from indirect pressure by edema or extravasation of blood into the injured soft parts surrounding the nerve. It is usually unilateral and is typically of the peripheral type, with the involvement of both the upper and the lower branches of the nerve. The paralyzed side appears smooth; the forehead cannot be wrinkled; the eye remains partially open; the corner of the mouth droops, and the nasolabial fold is obliterated. This type of paralysis is common, well understood and, in most instances, of a transient character.

Bilateral paralysis is most rare, because of the difficulty of applying the forceps in such a way as to damage the facial nerves on both sides. In such a case seen by Seeligmüller,[3] the forceps markings were plain on both sides of the head. Edgeworth [4] reported a case in a girl, aged 7 years, who from birth showed a bilateral facial paralysis of the peripheral type. Hearing and taste were normal, and there was no involvement of the palate. There was a definite history of bruising in front of both ears after forceps delivery, which led the author to consider this a case of bilateral facial paralysis due to forceps trauma.

---

1. Falloux: Les paralysies faciales congenitales, Thése de Paris, 1909.
2. Heller: Contribution a l'étude des paralysies faciales congénitales par agenése du rocher, Thèse de Paris, 1903.
3. Seeligmüller, cited by Moebius: München. med. Wchnschr. **39**:17, 41, 55, 1892.
4. Edgeworth, F. H.: Brit. M. J. **1**:11 (Jan. 6) 1894.

Rarely the paralysis may be of central origin, either directly from trauma to the facial area in the cortex or indirectly from hemorrhage into some portion of the upper neuron. Budin [5] reported a case in which the trauma of the forceps blade caused a large swelling over the posterior superior part of the right parietal, occipital, frontal and temporal regions, and on this side the inferior branch of the facial nerve was paralyzed. Undoubtedly the paralysis was due to pressure on the facial area in the cerebral cortex, probably from a fracture of the skull following the application of forceps. This cleared up in fifteen days. One of us (B.) has recently observed an instance of central facial paralysis due to hemorrhage in a boy, aged 3 years, who showed from birth partial paralysis of the inferior branch of the left facial nerve. During crying, especially, the mouth deviated to the right, while the left side remained immobile. The nasolabial groove was less prominent on the left. The child was the product of a difficult forceps operation. Associated with this was a diplegia of the lower extremities. Such complications are frequent with intracranial hemorrhage.

Facial Paralysis Due to Inherent Pressure Factors.—In this group the pressure does not come from obstetrical instruments, but either from forces exerted within the uterus during pregnancy, such as pressure from gross uterine content, causing abnormal posture of the fetus, or from pressure of the fetus during labor against certain rigid maternal prominences.

Antepartum Compression: Spontaneous facial paralysis resulting from intra-uterine pressure is associated with cranial and facial asymmetry. It is caused during the latter months of pregnancy by the head being pressed against the shoulder. This occurs in cases in which the intra-uterine pressure is greater than usual, such as might be found in first pregnancies with large babies or in multiple pregnancies. The manifestations are definite in most instances. The skin of the neck is thin, dry and sometimes excoriated; the parietal and temporal regions and lower jaw are flattened on the affected side; the ear is depressed, and the external auditory canal is narrowed. Proptosis is not uncommon. There is a peripheral type of paralysis of the facial nerve, as a result of this pressure. The cranial and facial appearance is often somewhat similar to that found in cases of wry neck or hemiatrophy, although in the latter the nerve is not involved, and certain abnormalities of the shoulder are not present. On roentgenologic examination the alveolar process is found to be flattened, and often the shoulder is depressed, together with bending of the clavicle. The pressure conditions causing this anomaly are well proved, and it is erroneous to attribute the cause

5. Budin, cited by Falloux (footnote 1, p. 99).

to nuclear atrophy or agenesis.[6] Occasionally asymetry of this sort is observed without the association of demonstrable nerve involvement. At present we have such a case under observation, one of twins with the aforementioned facial asymetry and a moderate degree of torticollis. Some of the so-called stigmas of degeneration are probably of this origin.

Heller reported several cases of congenital facial paralysis, characterized by facial paralysis of the peripheral type, with malformations of the ear, and especially the mastoid region. He attributed the paralysis to an agenesis of the petrous portion of the temporal bone, with resulting agenesis of the facial and auditory nerves, the external ear and the mastoid. However, as Essen-Möller [7] has pointed out, the bone and cartilage formation of the mastoid appears after development of the nerve. Therefore, the theory of a primary agenesis of the fallopian canal cannot hold. The occurrence of a secondary narrowing with resulting pressure atrophy or agenesis could explain this type of paralysis, for instance, the case of Geyl,[8] Levi,[9] and Rothschild, Marfan and Armand-Delille,[10] and possibly that of Soques and Heller.[11] If these cases cannot be explained in this way, they might be grouped under those due to agenesis of the nucleus, for in many such cases there are other developmental defects, especially about the ear. The fact that there is reasonable doubt as to the existence of this group of cases described by Heller and others makes it seem justifiable, for the sake of simplicity, to omit the subdivision. In dealing with a subject of this sort there are too many complex factors involved to put all cases showing deviations from the general groups into separate subdivisions. This tends only to becloud the issue. On review, Heller's cases do not conform sufficiently to one type to admit that there is such a distinct entity as he suggests.

In Geyl's case the new-born infant born of a short normal labor revealed a peripheral paresis of the right side of the face with some malformation, and underdevelopment of the cartilages of the tragus and concha of the right ear. No electrical reactions were reported. There is little evidence in this case to warrant the assumption that the cause of the paresis is agenesis of the mastoid. It could easily fit into the group caused by compression of the head against the shoulder or against some maternal hard part.

---

6. Abel: New-born Child with Congenital Peripheral Paralysis, Wien. med. Wchnschr. **77**:877 (June 25) 1927.

7. Essen-Möller: Ztschr. f. d. ges. Anat. **14**:52 (March) 1928.

8. Geyl, cited by Falloux: Centralbl. f. Gynäk., 1896, no. 24.

9. Levi, L., and Rothschild, H., cited by Falloux: Soc. méd. d. hôp., February, 1903.

10. Marfan and Armand-Delille: Paralysie Faciale Congénitale du Cote Droit, Soc. med. d. hôp. **18**:1007, 1901.

11. Soques and Heller, cited by Falloux: Soc. med. d. hôp., January, 1903.

The patient in the case of Soques and Heller, aged 6 years, the product of a full term pregnancy and normal noninstrumental delivery, showed a right facial paralysis of the peripheral type at birth. There was arrested development of both ears. On the left the external ear was malformed, and the external auditory canal narrowed. On the right, the affected side, the external ear was absent, and the canal was represented by a small opening. The malar bone and maxillary bone on the right were atrophic. The tongue and the palate were normal, and there was no difficulty of deglutition. The movements of the ocular muscles were normal. The electrical reactions were relatively normal for all the muscles but the frontals, which were diminished. This case could be included easily among the cases of nuclear agenesis, with accompanying malformation of the ears. As both ears were defective, and only the right facial nerve involved, there is no reason to attribute the paralysis to the ear defect. The underdevelopment of the ear, malar and maxillary bones on the side of the paralysis suggests the possibility of intrauterine posture playing some part in the development of the condition.

Levi and Rothschild reported the case of a new-born infant weighing 2,020 Gm. with a peripheral paralysis of the left facial nerve. Accouchement was normal. The left external ear was rudimentary, and the external auditory canal was represented by a dimple. The mastoid was normal. The ascending portion of the maxilla was defective, the zygomatic process making an anomalous articulation.

When the infant cried, the tongue deviated to the left. There was no malformation of the palate. The uvula deviated slightly to the right. There was slight diminution in the electrical reactions on the left side. The observations in this case are such that it takes little imagination to place it in the group of cases due to intra-uterine compression of the head against the shoulder or against some maternal bony part. The mastoid was admittedly normal, although the external ear and auditory canal were abnormal. The amniotic band found at the site of the ear defects is to be considered more as an associated bit of pathologic change than the cause of the facial paralysis and bony defects.

This leaves to be explained the case of Marfan and Armand-Delille, which has been included among those instances of agenesis of the mastoid. A girl, aged 3 months, born in normal labor, showed from birth a complete paralysis of the right side of the face, with absence of electrical excitability. The ocular movements were normal; there was no strabismus; sensation was normal; and movements of the extremities were normal. The external ear on the right side was rudimentary, the external auditory canal consisted of a narrow cul-de-sac, and the drum membrane was absent. The stylomastoid foramen and facial canal were absent, and the mastoid was aplastic.

At autopsy the root and nucleus of the facial nerve were found involved. The author called it secondary atrophy. From the observations one might place this case with the group due to intra-uterine compression, but the fact that the nucleus and nerve root were definitely involved brings up the possibility that here we are dealing with a primary agenesis of the nucleus with associated malformations of the ear. This case should not be considered one of primary agenesis of the ear.

Essen-Möller reported a case of right-sided peripheral facial paralysis, with a rudimentary left ear, absent auditory canal and multiple malformations of the shoulder, arm and thorax. It is interesting to note that the ear defects were on the side opposite the facial paralysis. This case fortifies the suspicion that the so-called mastoid agenesis has little to do with the development of facial paralysis. Apparently this case is simply one of agenesis of the facial nucleus with multiple malformations. In the case of Soques and Heller, both ears were also involved, although the paralysis was unilateral.

According to some authors, the malformation of the mastoid and external ear cannot be considered other than an incomplete formation in distant parts of the body, such as may be associated with congenital defects of motion in the field of cranial nerves. The part that local disturbances of nutrition play in development of such anomalies has not been sufficiently considered.

Intrapartum Compression: Spontaneous facial paralysis resulting from the trauma of labor usually follows extracranial pressure in the region of the facial nerve, especially in cases of protracted labor with large babies, contracted pelves or both. Certain maternal bony prominences such as the sacral promontory, the symphysis pubis, pubic rami, or ischial spines come into contact with the stylomastoid region of the infant during the strong uterine contractions of labor, with resulting injury to the facial nerve by direct pressure, edema or hemorrhage. Most of these follow anterior parietal presentations. Occasionally the pressure has been ascribed to violent uterine contractions alone.

Falloux in his thesis on congenital facial paralysis cited twelve such cases. Roulland,[12] Depaul,[13] Kennedy,[14] Geil,[15] Knapp,[16] Frank,[17] Vogel,[18] Stein,[19] Bonnaire[20] and Beriel.[21] The cases of Geil, Frank

12. Roulland, cited by Falloux: Thése de Paris, 1887.
13. Depaul, N., cited by Falloux: Thése de Paris, 1872.
14. Kennedy, cited by Falloux: Dublin Med. J., 1836.
15. Geil, cited by Falloux: Centralbl. f. Gynäk., July 4, 1896.
16. Knapp, cited by Falloux: Centralbl. f. Gynäk. July 4, 1896.
17. Frank, cited by Falloux: Centralbl. f. Gynäk., May 18, 1901.
18. Vogel, cited by Falloux: Ztschr. f. Geburtsh. u. Gynäk., vol. 48, p. 3.
19. Stein, cited by Falloux: Centralbl. f. Gynäk., March 18, 1905.
20. Bonnaire, cited by Falloux: Soc. d'obstet. de Paris, March 15, 1906.
21. Beriel, cited by Falloux: Rev. d. mal. de l'enf. 1906, p. 503.

and Beriel, which probably should not be considered in this group will be mentioned later. In one instance (Vogel), that of an infant delivered by Porro's cesarean section, the pressure was thought to come from crowding of the head against one of the pubic rami during the test of labor. Of this group, nine were cases of left-sided paralyses and two were right-sided; in one, the side was not mentioned. All of the patients with the exception of those in the cases of Beriel and Frank showed evidence of skull depressions or tumefactions from contact with some of the maternal bony prominences. Roulland's second case was probably due to pressure from the sacral promontory, Kennedy's the ischial spines, and Vogel and Stein's the horizontal ramus of the pubis. The condition in Knapp's and Roulland's second case was attributed to no especial bony prominence, but in both instances the pelves were greatly contracted. While such cases show evidence of marked intrapartum pressure by grooves, depressions or indentations of the skull of the affected side, the paralysis is probably due to edema or extravasation of blood in the region of the nerve. The lesions are unilateral, but total, and the paralysis in most instances is transient.

Geil's case was thought to be due to compression of the facial nerve by an amniotic band; if so, it should not be included in this group. It seems more likely that the case is due to compression in utero, and it should be classed among the group due to antepartum compression. In Frank's case the pressure was assumed to come from the shoulder of the fetus.

The case described by Beriel, which Falloux included among this group, is ostensibly one of antepartum compression. The cause of the paralysis in this case was attributed to the violent uterine contractions. The pelvis was normal, although the infant was large. Besides the total right-sided facial paralysis there were signs of pressure atrophy of the jaw on the right side, such as are found in the cases mentioned under antepartum compression. The symptoms persisted up to three months, after which they decreased, so that by the sixth month they had disappeared.

Fractures of the skull, especially the depressed variety, may cause facial paralysis of central origin by pressure of the depressed fragment on the facial area in the cortex, by hemorrhage about this region, or into the tract somewhere above the nucleus. The lower branch of the nerve is involved sometimes only in part. There are no electrical changes or atrophy. When the condition is due to hemorrhage, the paralysis usually appears shortly after birth, and is often complicated by other paralyses.

It is of importance to differentiate between the antepartum and intrapartum forms, because of the prognosis. Paralyses associated with

developmental defects of the ear, due to intra-uterine malposition and pressure, are more serious and the prognosis is poor. Those due to pressure of the head against maternal bony prominences during labor, are to be compared with the form of facial paralysis from forceps pressure, and like it, in these, the prognosis is usually good. Recovery is frequent.

In the cases of central paralysis due to fracture or hemorrhage recovery is not so likely. Especially is this true of the cases due to intracranial hemorrhage.

CONGENITAL FACIAL PARALYSIS OF SPONTANEOUS OR NONOBSTETRIC ORIGIN

Cases of spontaneous congenital facial paralyses of nonobstetric origin are those in which the nucleus and nerve roots are involved. In 1892, Moebius [22] reported forty-four cases of nuclear defects, two of which were his own. He called the condition "nuclear atrophy," and classified the diverse observations in the following way: (1) ophthalmoplegia externa with or without facial paralysis; (2) bilateral abducens and facial paralysis; (3) bilateral abducens paralysis; (4) bilateral oculomotor paralysis; (5) bilateral congenital ptosis; (6) congenital unilateral abducens paralysis; and (7) special form of congenital unilateral ptosis. This classification was further divided into the congenital form, and the more or less progressive form developing in childhood. There were only six cases of facial nerve palsy in this series, the first of which is of extremely doubtful origin (Recken,[23] Graefe,[24] Moebius,[25] Chisolm,[26] Harlan,[27] Schapringer [28]). In all of these the paralysis was bilateral, and all were associated with lesions of other cranial nerves; one with ophthalmoplegia externa (Recken); and five with abducens paralysis. In some, the facial involvement was complete; in others, partial on one or both sides. From time to time there have been reported other cases of bilateral facial paralysis, associated with involvement of adjacent cranial nerves, some of the earlier of which escaped the notice

22. Moebius, P. J.: Ueber infantilen Kernschwund, München. med. Wchnschr. **39**:17, 41, 55, 1892.

23. Recken: Zur Kasuistik und Lehre von den Augenmuskelläkmungen, Klin. Monatsbl. f. Augenh. **29**:340 (Oct.) 1891.

24. Graefe, A., cited by Moebius (footnote 22).

25. Moebius, P. J.: München. med. Wchnschr. **35**:6, 1888.

26. Chisolm: Paralysie congenitale de la sixieme et de la septiema paires chez un adulte, Arch. f. Augenh. **17**:414, 1887.

27. Harlan: Congenital Paralysis of Both Abducens and Facial Nerves, Tr. Am. Ophth. Soc. **3**:216, 1881.

28. Schapringer, A., cited by Moebius: Ueber angelborene beiderseitige pleuroplegia (Abducens und Faciallamung), Med. Monatschr. **12**:622, 1889.

of Moebius; thus the cases of Foville,[29] Mauthner,[30] Block,[31] Fryer,[32] Procopovici,[33] Huebner,[34] Batten,[35] Gutzmann,[36] Gierlich,[37] Fry,[38] Cadwalader [39] and Kerby [40] involved both the facial and the abducens nerves. Falloux mentioned two cases of Gierlich, but the two references concern the one case. The case reported by Procopovici has been erroneously reported by some as an uncomplicated facial diplegia. Bilateral facial paralysis with ophthalmoplegia externa has been reported by Bach,[41] Gesepy,[42] and Decroly,[43] the latter's patient also showing difficulty in deglutition. Schmidt's [44] case was one of bilateral facial and abducens paralysis with involvement of the hypoglossal nerve on the left side, more than on the right.

We have discarded several cases reported by others as being of congenital origin. Uthoff's [45] case of bilateral facial paralysis with ophthalmoplegia externa on careful scrutiny turned out to be one of generalized tuberculosis. The case reported by Julius Stein,[46] one of facial diplegia with paralysis of the hypoglossal nerve, cannot be included, for the condition was associated with a generalized progressive muscular paralysis, possibly of the type described by Londe, Brissaud and Marie.

So many cases of unilateral facial paralysis of nuclear origin have been reported that no attempt will be made to list them. For excellent

---

29. Foville, A., cited by M. P. Sainton.

30. Mauthner, cited by Recken: Die nicht nuclearen Lähmungen der Augenmuskel, 1885, p. 432.

31. Block, cited by Thomas, H. M.: Thesis, Berlin, 1891.

32. Fryer, cited by Thomas, H. M.: Case of Bilateral Congenital External Ophthalmoplegia and Congenital Facial Paralysis, Ann. Ophth. Otol. Kansas City, 1892, p. 82.

33. Procopovici, cited by Thomas, H. M.: Sur la paralysie congénitale bilaterale de l'abducens facialis, Arch. f. Augenh. **34**:44, 1896.

34. Huebner, O.: Ueber angeborenen Kernmangel (Infantiler Kernschwund Moebius), Charité-Ann. **25**:211, 1900.

35. Batten, F. E.: Congenital Affection of Sixth, Seventh and Twelfth Cranial Nuclei, Brain **28**:359, 1903.

36. Gutzmann, cited by Falloux: Soc. de méd. int. de Berlin, Nov. 20, 1905.

37. Gierlich: Ueber infantilen Kernschwund, Arch. f. Kinderh. **46**:442, 1907; Deutsche med. Wchnschr. **31**:1462 (Sept. 14) 1905.

38. Fry, F. R., and Kasak, M.: Congenital Facial Paralysis, Arch. Neurol. & Psychiat. **2**:638 (Dec.) 1919.

39. Cadwalader: A Clinical Report of Two Cases of Agenesis of the Cranial Nerves, Am. J. M. Sc. **163**:744, 1922.

40. Kerby, D. B.: Arch. Ophth. **52**:452, 1923.

41. Bach, cited by Thomas: Centralbl. f. Nervenheilk. **16**:57, 1893.

42. Gesepy, cited by Thomas: Arch. d'opht. **14**:273, 1894.

43. Decroly, cited by Falloux: J. de neurol. 1902, no. 23.

44. Schmidt, cited by Thomas: Deutsche. Ztschr. f. Nervenh. **10**:400, 1897.

45. Uthoff: Tageblatt der 59 Naturforsch. Versammlung, p. 155.

46. Stein, J., cited by Sainton: Arch. f. klin. Med. **6**:593, 1869.

bibliographies the articles of Falloux,[1] Thomas,[47] Kunn,[48] Moebius[22] and Sainton[49] should be consulted. The extent of involvement varies considerably as does the association with other cranial nerve lesions. The uncomplicated cases are in the minority.

Moebius stated that bilateral congenital facial paralysis unassociated with paralysis of the sixth cranial nerve was unknown to him. The nuclei of these two nerves are situated so close to one another in the pons that it would be difficult, indeed, for one to be affected without the other. Kunn asserted that bilateral facial paralysis was accompanied by involvement of other cranial nerves, while the unilateral form was often uncomplicated. While, in general, these statements are true, a critical review of the literature reveals a few cases in which the nucleus of the seventh nerve only has been involved. We are in accord with Thomas'[47] assertion that his two cases in brothers are the first authentic ones on record.

REPORT OF CASES

Case 1 (Thomas).—A man, aged 21, had from birth shown signs of bilateral facial paralysis. The labor had been normal. He had never suckled well, the face was at all times expressionless and the eyes could not be closed. The mouth was held partly open, the lips were everted and efforts at elevating the upper lip or puckering the mouth were unsuccessful. The mouth was drawn somewhat downward to the right. The eyebrows could not be elevated nor the forehead wrinkled. On attempting to close the eyes, the lids became relaxed and the eyeballs rolled upward. Enunciation was indistinct. Sensation, taste and mastication were all normal. The soft palate was normal and the pharyngeal reflex present. Deafness in both ears was attributed to measles. The eyes could be moved freely and equally in all directions. The lobe of the left ear was malformed.

Case 2 (Thomas).—This man, aged 19, showed from birth a masklike face, with partially open eyes and mouth. The lips could not be moved, and nursing had been performed by use of the tongue. The forehead could not be wrinkled, nor could the eyes be closed. Speech was imperfect due to inability to use the lips. Mastication, sensation and taste were normal. There was partial deafness in both ears. The ocular movements were normal. The lobe of the right ear was notched.

Case 3 (Taylor[50]).—A boy, aged 15, had never shown any facial expression. There was no movement of the face on crying. The labor had been noninstrumental, and there was no history of otorrhea. At the age of 5 both legs were said to have become paralyzed, but recovery soon took place. Examination showed a complete bilateral facial paralysis save for slight voluntary power in the risorius, the depressor of the angle of the mouth and the orbicularis oris; otherwise the face was completely masklike. The palpebral fissure was never closed completely, and the mouth always gaped. The child could talk and could be understood, although the labials were not pronounced. The soft palate moved sluggishly, and

47. Thomas, H. M.: J. Nerv. & Ment. Dis. **25**:571, 1898.

48. Kunn, cited by Thomas: Beitr. z. Augenh. **19**:1, 1895.

49. Sainton, M. P.: Les diplegies facialis totales, Gaz. d. hôp. **74**:132, 1901.

50. Taylor, S.: A Case of Bilateral Facial Palsy, Tr. Clin. Soc. London **35**: 177, 1902.

there was slight difficulty in swallowing. No other palsies were noted. Electrical reactions were not determined. From the photographs one noted that the nasolabial folds were not completely obliterated and the eyeballs rolled upward on an apparent attempt to close the lids. Ptosis was present but not so marked as in some cases. There were no ear defects.

Case 4 (Rainy and Fowler [51]).—A girl, aged 10 weeks, showed complete paralysis of both seventh nerves. The face was practically motionless and devoid of expression when the child cried, save for a contraction of the fibers of the occipitofrontalis just above the extreme inner angle of the right eye, and of the right depressor anguli oris. The upper lip protruded somewhat, while the lower one was drawn slightly downward and toward the right side. The lips were motionless, and the lids could not be closed tightly, especially on the left side. The ocular movements were normal in all directions, and there was no other paralysis. Taste was present, but it was uncertain whether or not it was over the whole tongue. Swallowing was well carried out. There was no response to the faradic and galvanic currents, except on one examination when the masseters and orbicularis palpebrarum responded feebly. The child died of bronchopneumonia shortly after admission. Although this child was born of instrumental labor, the autopsy report showed the paralysis due to an atrophy of the nucleus and nerves. The muscle fibers were normal.

Case 5 (Fry [52]).—An infant, aged 11 months, showed paralysis of the lower right side of the face, with paresis of the upper right side and the entire left side. The mouth remained partially open, and the lower lip was everted. The eyes remained slightly open and could not be completely closed. On crying there was evident lack of mobility of the upper half of the face, as well as the lower, more so on the right than on the left. The condition was noticed the day after birth, when it was observed that the infant could not nurse well.

Case 6 (Cadwalader [20]).—A girl, aged 13 years, showed a bilateral facial paralysis from birth. She was born after normal labor. As an infant, nursing was difficult because of lack of use of the facial muscles, especially those of the lips. Hearing was impaired on both sides. Vision and ocular movements were normal. Mentality was normal.

Case 7 (Authors' Case).—*History.*—Max R., aged 9 months, was brought to a welfare clinic for examination because the mother noticed that the child was not normal. He has been seen frequently since that time, and at the age of 15 months was sent into the hospital for observation. He is the second child in the family. The older brother, aged 3 years, is normal.

The mother had not felt well during her second pregnancy, and said that the membranes had ruptured three weeks before labor began. Delivery occurred after a noninstrumental dry labor of twenty-four hours' duration, and the full term baby weighed approximately 8 pounds (3.6 Kg.) at birth. The baby breathed spontaneously after birth, did not cry loudly, and seemed lethargic during the first three days. No convulsions were noticed. For the first three days the infant would not nurse and was fed with a medicine dropper. The mother stated that at no time did she feel the infant's tongue against her nipples during nursing. The

---

51. Rainy, H., and Fowler, J. S.: Congenital Facial Diplegia Due to Nuclear Lesion, Rev. neurol. **1**:149, 1903.

52. Fry, F. R.: Congenital Double Facial Paralysis, Interstate M. J. **25**:856, 1918; Congenital Facial Paralysis, J. A. M. A. **14**:699 (June 19) 1920.

infant was nursed for fourteen months, as it would never suckle a rubber nipple. The mother thought the baby could swallow fairly well. The mouth had always been partially open.

The family history was negative, and there was no history of any similar condition in either of the parent's families.

A circumcision was performed on the tenth day. During the administration of ether the infant stopped breathing, but after considerable difficulty it was resuscitated. At the age of 2 months it was operated on for clubfoot, and a cast was applied. When 3 months old, the baby developed whooping cough and measles. The child had frequent colds; there had been a tendency for the eyes to water easily, and a purulent lacrymal discharge from the eyes had been noticed occasionally. The baby cut its first teeth at about 6 months of age, sat up at 10 months,

Fig. 1.—Appearance of child while crying.

crawled at 13 months, stood up at 14 months and could walk with the aid of a walker at 15 months. He now can walk unaided. Throughout the first nine months the mother thought the baby was subnormal mentally, but at present she feels that it is normal.

The present complaint—lack of expression of the face—was noticed by the mother in the first few days after birth, but she could not state whether or not it was present at birth. The facial muscles have never been seen to function, the face always remaining expressionless. Once when the child was laughing harder than normal, the mother noticed a dimple in the cheek, and occasionally there seems to be a slight twitching of the muscles at the corner of the mouth.

The infant has been examined frequently, but the results have been essentially the same each time.

Examination.—When first seen at the age of 9 months (August, 1927), the child appeared fairly well nourished. It looked stupid, with an expressionless, masklike face, drooping eyelids, sagging lower jaw, partially opened mouth, and

depressed corners of the mouth. The face was free from wrinkles, and there were no nasolabial folds on either side. When the child cried or laughed, there was no discernible movement of the muscles of the face. The forehead did not wrinkle. Figure 1, which shows the child while crying, could not be differentiated from figure 2, taken during repose. The only noticeable difference was in the color of the skin, which during crying became vividly flushed. At the same time the vessels of the neck became distended, much more so than one usually observed in the normal infant. The lids drooped and the head was held backward somewhat to compensate for this (fig. 1). The inner epicanthic folds were prominent. The lids blinked feebly, and the eyeballs rolled upward when an object approached the eyes. The pupils reacted to light and in accommodation, and the ocular movements were normal. It was difficult to get the child to look upward because of the

Fig. 2.—Appearance of child during repose.

ptosis. The eyeballs rolled upward when an attempt was made to close the eyelids. There were no malformations of the ear. The root of the nose was broader than normal, and the bridge was developed normally for a child of this age. The corners of the mouth drooped and the jaw sagged. This is well shown in figure 3. The tongue moved actively from side to side, and could be protruded normally. When the child was given a nipple, the lips did not close about it. The palate was high, and the throat and tongue were normal. Eight teeth were present (15 months). Hearing, taste and smell were apparently normal. The forehead was fairly well developed, and the contour of the head was somewhat tower-like (fig. 3). The body was moderately well developed. The tissue turgor was subnormal, and the muscles were flabby. The chest was barrel-shaped with the usual flaring at the lower border, found in rickets. There was moderate beading of the ribs and thickening of the epiphyses of the bones of the forearms and legs. There was definite roentgenologic evidence of rickets.

The thyroid gland could not be palpated. Many small cervical glands were felt. No thymic dulness was demonstrated. The lungs and heart were normal. The liver was 1 cm. below the costal margin. The spleen was palpable. The abdomen was somewhat prominent, but was otherwise normal. The extremities were normal save for the previously noted left equino varus. The fingers were held in a peculiar pill-rolling position (figs. 1, 2 and 3). The reflexes, save those of the face, were normally active. The electrical reactions to the galvanic current were absent until the current was increased to 30 milliamperes, when there occurred contractions of both the upper and the lower groups of facial muscles. The child's mentality was within normal limits, although on the first examination the infant appeared to be subnormal, due to the facial expression.

The laboratory observations were essentially normal. Examination of the blood showed: erythrocytes, 3,800,000; leukocytes, 8,000; hemoglobin, 70 per cent

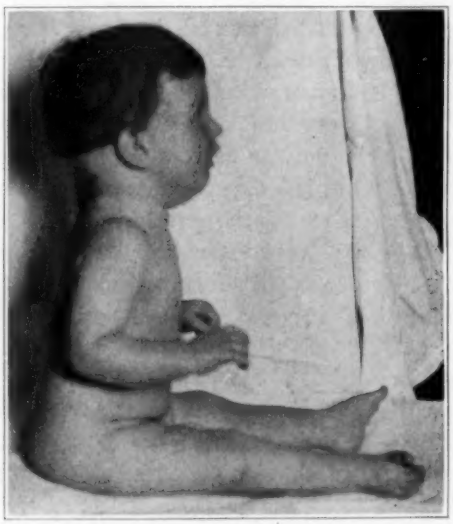


Fig. 3.—Profile of child while crying.

(Dare); the differential cell count was not remarkable; and the Wassermann reaction of the blood was negative. The spinal fluid was clear and under normal pressure; the Wassermann reaction of the spinal fluid was negative, and the colloidal gold curve was normal. The urine was normal.

On examination at the age of 34 months, a few changes were noted. The lids were closed during sleep and the blinking movements were improved to such an extent that there was no epiphora. Hearing was definitely acute. Enunciation was imperfect as far as the use of labials is concerned. The mentality was normal for a child of this age. Questions were answered readily.

To summarize, this case is one of complete bilateral facial paralysis, evidently due to involvement of the nuclei of the seventh cranial nerves, since the extracranial portion of the facial nerves and muscles function as demonstrated by the use of a strong galvanic current. There is no apparent involvement of any other cranial nerves. Associated with this paralysis is a unilateral equino varus. The condition has remained practically unchanged during the observation period of over two years.

A few other cases have been reported, but after careful investigation they cannot be included. The case reported by Collins,[53] that of a girl, aged 7 years, from whom no history could be obtained, revealed a complete bilateral facial paralysis. However, the fact that the extensors of the legs were paralyzed, together with the lack of history, makes it necessary to omit this case. The chances are that it is an instance of acquired paralysis. In Procopovici's case, the sixth pair of nerves was involved, although some of the reports fail to mention this fact.

COMMENT

As far as we have been able to determine, only seven cases of congenital bilateral facial paralysis uncomplicated by other cranial nerve involvement have been reported. In all but one (Fry), the paralysis was complete. In two (Thomas) the condition was familial, and there were slight ear defects. In the authors' case there was an associated left equino varus. In all of the cases the condition was stationary from birth.

Moebius attributed the nuclear defect to atrophy, chiefly because in some of his cases, those in which the symptoms appeared some time after birth, there was progression of the paralysis. This occurs in cases developing in childhood, making it difficult early to differentiate this condition from some of the progressive forms of infantile paralysis. Infantile familial progressive bulbar palsy (Londe) appears late, progresses rapidly with regional extension, that is, involvement of other cranial nerves, especially those of deglutition. It is always fatal. Infantile pseudobulbar palsy (Oppenheim) usually affects the fifth, seventh and twelfth cranial nerves, the lower facial more than the upper, and is always bilateral. It is apparent that differentiation from nuclear agenesis is difficult, so much so that it is conceivable that the two conditions are merely mild variations of the same disorder. The explanation of the progression mentioned in some of Moebius' older cases can be found on the assumption that there is a lack of ability of the involved tissues to carry on the necessary reparative processes of life, that is, there is defective vital endurance of the tissues—abioatrophy. If by chance the nuclei are underdeveloped to begin with, it seems justifiable to assume that the continued demand on the tissues to carry on work they are unequal to would result in atrophy. This idea of so-called lack of vital endurance has been used to explain other progressive nervous diseases, such as infantile familial progressive bulbar paralysis and amaurotic family idiocy.

However, the primary process in these nuclear defects is probably an agenesis or partial agenesis as Kunn suggested shortly after the appear-

53. Collins, E. T.: Case of Double Facial Paralysis, Tr. Ophth. Soc. U. Kingdom **35**:371, 1915.

ance of Moebius' reports, and as many others have since concluded. At the present time the term "nuclear agenesis" is the one chosen for this condition rather than the "nuclear atrophy," or "Kernschwund" of Moebius.

The pathology of the condition has been studied by Heubner and others. The observations are essentially those of agenesis of the nuclei and nerve roots. The cells of the nucleus are absent or diminished in number, and those remaining are abnormally small. The nerve roots are small or absent, and in some instances the trunk is missing or of diminished size. These changes may be unilateral or bilateral. Histologic preparations of the case studied by Rainy and Fowler showed atrophy of the nucleus and roots and trunk of the facial nerve. No other nuclei were involved. These results are contrary to those usually observed. The authors conclude that through lack of inherent vitality or defective nutrition the cells perished, leaving evidence of their former existence in degenerated nerves.

The etiology of the condition is obscure. Many suggestions have been made, but none have been accepted. Alcoholism and syphilis play no part. The history of antenatal disease or weakness is not common. A history of other forms of nervous disease is frequently obtained, and in some instances the condition is familial. The two patients observed by Thomas were brothers, and in Cadwalader's second case a brother was similarly affected. Trautman [54] reported six cases in one family. Thus, while there may be a familial tendency in some cases, the condition cannot be regarded as hereditary. The usual attempts to incriminate toxins, fetal inflammations and encephalitis have been unsuccessful. The fact that many of the cases reported have been associated with other malformations might suggest the possibility of a nutritional defect being the causative factor in all these conditions.

The diagnosis is made on the discovery of lack of function of the facial muscles at birth and the exclusion of other causes of seventh nerve involvement. The prognosis regarding life is good, but as far as improvement is concerned, little can be expected. Treatment is unavailing.

SUMMARY

For simplicity's sake congenital facial paralysis may be divided into two large groups: those of obstetric origin, and those of spontaneous or nonobstetric origin. The first class may be further subdivided into paralysis from forceps trauma and paralysis due to inherent pressure factors such as antepartum compression or intrapartum compression.

54. Trautman, D.: Beitrag zur Kasuistik kongenitaler Facialisparesen, Ztschr. f. de ges. Neurol. u. Psychiat. **100**:289, 1925-1926.

The cases due to forceps trauma are usually unilateral, although bilateral cases have been observed. Likewise, while they are usually peripheral, central paralyses are known. The prognosis is favorable.

The cases due to antepartum compression of the head of the fetus against its own shoulder, or against maternal bony prominences, manifest along with unilateral peripheral facial paralyses certain pressure defects of the ear, face, jaw and shoulder. The prognosis in these cases is unfavorable.

The other group, caused by intrapartum compression, shows unilateral peripheral facial paralysis together with evidence of injury in the region of the nerve, such as edema, extravasation of blood and depressions in the skull. Occasionally a central type of paralysis is observed. The prognosis in this group is usually good.

The second class, those of spontaneous or nonobstetric origin, is thought to be due to agenesis of the nuclei of the seventh nerve, with similar involvement of the root and sometimes of the trunk of the nerve. The symptoms vary according to the extent of involvement from total bilateral to partial unilateral paralysis. Other cranial nerves, especially the sixth and twelfth, are frequently involved. Cases of uncomplicated bilateral facial paralysis of nuclear origin are extremely rare. Six such instances are reviewed, and another is reported by the authors.

718 Boston Building.
Copper Building.

JUVENILE FLAT FEET *

H. C. SCHUMM, M.D.
MILWAUKEE

In any discussion of flat feet, one must first appreciate what is meant by a flat foot. It is not necessarily a foot in which the "arches are lost or flattened out," as is the popular conception.

For many years Whitman [1] has been preaching the doctrine that the most direct of the predisposing causes of so-called flatfoot is a posture that simulates the deformity which may be defined as a persistent attitude of abduction. It is now believed that this simulating posture is actually the first stage of flatfoot.

Morton [2] has aptly brought out the fact that it is the position of the os calsis as a means of regulating "balance" in the distribution of body weight that is of prime importance in the foot. The muscles of the foot are not weight-supporting but serve to balance the body weight on the foot or more particularly on the os calsis. Any derangement in this delicate balance results in an improper transmission of the body weight to the supporting structure.

The prevalence of flat feet was first forcibly brought to the attention of the world during the World War, when it is estimated that from 30 to 40 per cent of all men examined by the exemption board during 1917 presented either potential or actual disability, the result of flat or weak feet. These are reports in which the percentage of flat feet found has been as high as 50 per cent. Brahdy [3] reported that 19 per cent of a large number of children of preschool age who reported for routine examinatio were flat footed. Such startling statistics on the prevalence of flat feet naturally cause inquiry as to the cause. Among the etiologic factors usually given are: muscle weakness, improper footwear, systemic disease, trauma, excessive weight, developmental defects and congenital anomalies.

---

* Submitted for publication, Oct. 14, 1929.

* From the Milwaukee Children's Hospital.

1. Whitman, Royal: An Analysis of the Weak Foot, Med. Rec. **94**:49 (July 13) 1918.

2. Morton, Dudley J.: J. Bone & Joint Surg. **6**:368 (April) 1924.

3. Brahdy, M. Bernhard: Flat Feet in Children, Arch. Pediat. **44**:86 (Feb.) 1927.

The pathologic anatomy of flatfoot has been well brought out by Brahdy. He stated, "We are dealing essentially with an abducted and everted foot in which certain muscles and their tendons (tibialis anterior and tibialis posterior) are stretched and lose their elasticity, while other tendons and ligaments (lateral external) are contracted and shortened. A gradual separation or change in relation between some of the mesial bones of the foot takes place and the arches fall."

According to the mobility of the foot, flatfoot disorders may be divided into two stages: (1) functional deformity during which the false load (improperly transmitted body weight) predominates as the destructive agent and which tends progressively to break down the integrity of the foot by causing increased disorders in its normal mechanics, and (2) the stage of structural deformity with actual bone distortion. The former is naturally the best stage for treatment.

Until recent years, the presence of flat feet among children was seldom recognized clinically, unless it was a case in which the deformity was marked or when the child complained of pain. Because of this fact, if treatment was attempted, it was started so late that the results were not what they should have been.

A fact that is of most significance and interest to me is that after careful examination and investigation, I found just as high a percentage of the so-called potential flat feet among new-born infants as of actual flat feet among school children or as was found in the examinations by the draft boards.

Among 200 new-born infants recently examined by me, 29 per cent showed the typical position of abduction and eversion when the foot is at rest. This typical flatfoot position may be the result of either continued intra-uterine pressure (Spitzy [4]) or of fetal arrest in the growth of the epiphysis, as believed by Böhm.[5]

During the past few years, it has become evident that more physicians and many more parents are recognizing flat feet at an earlier age. Many more children between the ages of 3 and 8 are being placed under observation and treatment than formerly. Most of them are examined long before they have actual symptoms, which is most encouraging.

The most diagnostic sign is the presence of abduction of the heel, and in more pronounced cases, eversion of the fore foot. This causes a prominence of the medial border of the foot in the region of the scaphoid bone. This pronation and eversion is usually present when the child is at rest, but it is more pronounced when the child is standing

4. Spitzy, in Pfaundler and Schlossman: Diseases of Children, Philadelphia, J. B. Lippincott Company, 1912.

5. Böhm, to be published in J. Bone & Joint Surg., January, 1930.

in his bare feet. The actual presence or absence of an "arch" in the foot is of no importance. Weak flat feet may show good arches. Conversely, good feet may show none, because of the fat pads which are so commonly present up to the age of 4 or 5 years. A common symptom, especially in the small child, is the tendency to toe in or walk pigeon-toed. In most cases this is a protective mechanism by which the strain on the stretched and tired muscles and ligaments is relieved. This strain is usually quickly relieved by treatment. When untreated, it seldom persists after the child is 5 or 6 years of age. If the flat feet are more pronouced the child may stand with his toes turned outward. In such a case there may be a shortening of the achilles tendons. When the outer borders of the heels are worn away and the child has flexible feet, it is usually a sign of flat feet. The inner border of the soles may be worn away in the more advanced cases.

Children with flat feet tire easily. They sometimes complain of pain and they often want to be carried. It is normal for children to be active and want to play and never be conscious of their feet.

The earlier in infancy that flat feet are recognized, and the earlier treatment is instituted, the more satisfactory will be the results. The family physician or pediatrician, who is the first observer, should be on the watch for the condition. (Exaggerated cases should be referred to an orthopedic surgeon.) The pediatrician can well treat the children with mild cases seen in early infancy. Manipulation of the feet into adduction and inversion fifteen times, three times a day, is often sufficient. In the more advanced cases the application of light splints or plaster casts during the first months of life is often all the treatment that may be necessary.

As prophylactic measures, the following rules should be observed:

1. Never force nor press feet of small children into shoes.
2. Encourage creeping, but discourage abnormal locomotion, such as sliding over the floor on the buttocks.
3. Never encourage small children to walk. Let them walk only when they want to of their own accord.
4. Never take children for long walks, when there is little or no opportunity to rest when fatigued.

The choice of shoes is of primary importance in the prevention and treatment of the condition. Before a child begins bearing weight, a roomy shoe of soft flexible kid with an unresisting sole is desirable, because it allows free motion of the foot. As soon as the foot is used in weight-bearing for any length of time, a more substantial shoe is indicated.

According to Adams,[6] seven points are essential for a well fitted shoe. These, however, are too detailed to describe here. The essential points are:

(1) A fairly stiff sole

(2) A shoe broad enough across the toes

(3) Sufficient length not to cause pressure against end of the toes, so that the breadth of the finger can be placed between the end of the toe of the shoe and the toe of the child

(4) Sufficient height across the toe and vamp; the moccasin type of shoe is preferable.

As soon as the child is walking, the inner border of the heel should be raised from one-eighth to one-fourth inch and flared inward from nothing to one-fourth inch, depending on the degree of pronation. These altered heels should be worn from two to eight years. In more pronounced cases, felt pads, giving support beneath the head of the astragalus, or even celluloid foot plates may be indicated. Massage and occasionally contrast baths are beneficial. Very young children may be taught to walk on a doubled inclined plane. Older children may be given adduction or supination exercises fifteen times, three times a day.

SUMMARY

1. The potential flatfoot may be recognized at birth.

2. An early diagnosis should be made and treatment begun at once.

3. Mild cases are benefited by exercises, massage and heel alterations to correct the axis of weight-bearing.

6. Adams, John P.: Proper Shoeing of the Child, J. A. M. A. **92**:1753 (May 25) 1929.

Pediatric Biographies

J. STARSMARE
1664

JOHN RUHRÄH, M.D.
BALTIMORE

There is an interesting book on the diseases of children by J. S., which was printed in 1664. My own search for the identity of J. S. was fruitless. One opinion was that the book was written by Jane Sharp, a mythical midwife, under whose name some one, in 1671, published "The Compleat Midwife." Recently, Dr. Ernest Caulfield, of Hartford, Conn., called my attention to the fact that in "A Transcript of the Registers of the Worshipful Company of Stationers, 1640-1708" (London, 1913 vol. II, p. 333), there is the following:

Nov. 13, 1663

Entred . . . under the hands of Master Roger Le Strange and Master Luke Fawne, warden, a booke or coppie intituled ΠΑΙΔΩΝ ΝΟΣΗΜΑΤΑ; or childrens deseases, both outward and inward, from the time of their Birth, to fowerteene yeares of age. With their natures, causes, signes, presages and cures, In Three Bookes. 1– Of External deseases. 2- Of Universall deseases. 3- Of Inward deseases. Also the Resolucon of many profitable questions concerning childeren and of nurses and of nursing children, by J. Starsmare, Physician. . . . .

J. S. apparently lived at Oundle, in Northhampton. My informant also states there is a record of a similar name among the Oxford graduates about the time that J. S. lived.

The book is chiefly of interest as showing what was in vogue in pediatric practice in the time of Charles the second.

One short extract must serve as an illustration. Note the prognosis based on changes in sensation.

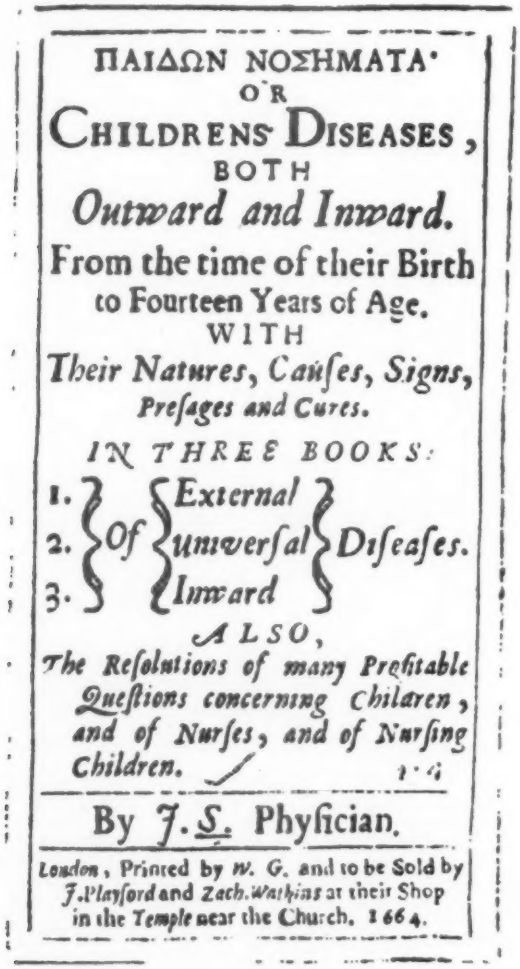
The chronic paralysis without changes in sensation being due to irreparable lesions, and paralyses with numbness or loss of sensibility being cases of neuritis, they tended to spontaneous cure so that the doctor and whatever remedy happened to be chosen got the benefit.

A Numness and Palsy, is a distemper of the same kind, and differ only in degree, for a Numness is but as it were an imperfect Palsy, and a Palsy is as it were a certain great Numness.

The cause is a pituitous humour which by coldness & moistness thickens & obstructs the Nerves, that they cannot receive the animal Spirits, or Influence of the animal faculty; & as this obstruction and condensation of the nerves and ways of the faculties is more or lesse, so it makes numnesse or weker motion or want of motion. In a *Palsy* there is no motion because the wayes of the faculties are

totally stopt, in a numnesse they are not stopt completely, therefore the faculty operates, and there is a weak motion.

The external causes are compressions by falls, blowes, binding tumours or other causes which can compresse and condensate the nerves that a free ingresse of the faculty is hindered.



ΠΑΙΔΩΝ ΝΟΣΗΜΑΤΑ·
OR
CHILDRENS DISEASES,
BOTH
Outward and Inward.
From the time of their Birth
to Fourteen Years of Age.
WITH
Their Natures, Cauſes, Signs,
Preſages and Cures.
IN THREE BOOKS:
1. 2. 3. } *Of* { *External, univerſal, Inward* } *Diſeaſes.*
ALSO,
The Reſolutions of many Profitable Queſtions concerning Children, and of Nurſes, and of Nurſing Children.
By *J. S.* Phyſician.
London, Printed by *W. G.* and to be Sold by *J. Playford* and *Zach. Watkins* at their Shop in the *Temple* near the Church. 1664.

Title page of work by "J. S."

Signs, if a part is affected with the *Palsy* it cannot move, and is called the *Dead Palsy;* if with numnesse, the party can move but weakly and with difficulty. If the Disease be in the *Spinalis medulla* the Arms and all the inferiour parts are hurt; if the right part of it is affected, all the parts on the right side, if the left, the left parts. If in the *Osse sacro* the parts above it are well and the parts inferiour to it are hurt.

Prog. every *Palsy* especially that which is inveterate is difficult to cure in Children but numnesse is more easie to cure.

If a *Feaver* or trembling comes upon a *Palsy* or numnesse it is very helpfull, for the *Feaver* dissolves the matter of the Disease and the trembling drives the same matter from the nerves.

The older writers, through either accident or design, got something picturesque into their descriptions. If one has a liking for fine phrases and an ear for words, great pleasure is to be derived merely from the joy which these old fellows evidently got from their own writing. Examples are numerous and the following paragraph on worms is in point.

The Signs of the three sorts of Worms in common are many, a Stinking breath, unquiet sleep, with starting, trembling, grating of the Teeth, Itching, and often rubbing of the Nose, paleness of the Face, red by Intervals, the Eyes hollow and darkish, the white whereof being turned pale or yellow, spitting much Phlegme, the swelling of the belly, with murmuring and noise in it, the Griping of the Belly which is worse, when one is fasting, sometimes looses, Vomiting and Epilepsy.

The Signs of the particular Worms; if they be long, then the biting of the Belly is more vehement, a little drye Cough, Hiccough, a vain desire of Vomiting, abominating meat, sounding, troublesome dreams, with trembling, rising up, and crying out.

News and Comment

SOCIETY NEWS

The Second International Pediatric Congress in Stockholm, August 17—August 20, 1930.—After having conferred with, and received the promise of support from, leading pediatricians in practically all the countries of the world, the Swedish pediatricians have decided to issue invitations to an international pediatric congress in Stockholm, to last from August 17 to and including August 20, 1930.

Notification of lectures is to be made before March 1, 1930, at the latest.

The program of the conference will, if necessary, be carried on in separate groups.

It is desirable that those wishing to attend signify their intention of doing so as soon as possible, so that the approximate number to be expected may be calculated, and arrangements made in good time for their housing and comfort. This is of great importance because of the Swedish Art and Industry Exposition which will be going on in Stockholm during the summer of 1930.

The admission fee to the conference is twenty Swedish crowns (Kr. 20).

All communications are to be addressed to *The Second International Pediatric Congress, Stockholm, Sweden.* Telegraphic address: *Pediatric, Stockholm.*

Applications for tickets of admission should preferably be accompanied by the fee, sent as a check or money order.

For the avoidance of errors, it is absolutely necessary that all names and addresses be written with printed characters or typewritten.

Further announcements will appear later on.

Stockholm, September, 1929.

J. JUNDELL, M.D.,
Professor of pediatrics, committee chairman

KJ. O. AF KLERCKER, M.D., professor of pediatrics, Lund.

R. NORDGREN, M.D., chairman of the pediatric section, Swedish Medical Association, Stockholm.

I. THORLING, M.D., professor of pediatrics, Uppsala.

WILH. WERNSTEDT, M.D., professor of pediatrics, Stockholm.

NILS MALMBERG, M.D.,
Lecturer in pediatrics, Stockholm, committee secretary.

University of Michigan Pediatric and Infectious Disease Society.—There was a two day meeting of the society on Nov. 8 and 9, 1929, at Ann Arbor, Mich. The program included a forum on meningitis, rheumatic diseases and the treatment for diarrhea.

GENERAL

International Pediatric Congress.—The committee for the United States for the Second International Pediatric Congress at Stockholm, August 17 to 20, 1930, is composed of the following members: Kenneth D. Blackfan, Boston; Henry Helmholz, Rochester, Minn.; A. F. Hess, New York; J. H. Hess, Chicago; William Palmer Lucas, San Francisco; Hugh McCulloch, St. Louis, and Edwards A. Park, Baltimore. The chairman is Clifford G. Grulee, Chicago, and the secretary, C. A. Aldrich, Winnetka, Ill.

The committee will pass on the qualifications of men and essays from this country to be presented at the meeting. As the subjects for discussion have not yet been definitely determined, further announcements will be made in this journal.

Bas-Relief of Dr. Howland Unveiled.—On Oct. 17, 1929, in the amphitheater of the Harriet Lane Home for Invalid Children of Johns Hopkins Hospital,

Baltimore, a bas-relief of Dr. John Howland was unveiled. President Ames made a brief introductory address, but that of Dr. Thayer was the principal address of the day.

Lectureship Founded in Honor of Dr. Blackader.—In recognition of the eighty-second birthday of Dr. Alexander D. Blackader, and in appreciation of his long service as editor of the *Canadian Medical Association Journal*, the Canadian Medical Association will establish a lectureship in diseases of children in his honor.

UNIVERSITY NEWS

Dr. John Bokay, who has been Professor of Pediatrics and head of the Department of Pediatrics at the University of Budapest for over thirty years, has retired under the law of age limit.

Dr. Paul Heim, who had just been appointed Professor of Pediatrics at the University of Budapest to succeed Dr. Bokay, died suddenly on October 23. Hungarian pediatrics lost one of its most able representatives in Dr. Heim.

The University of Chicago has received $20,000 from Dr. Walter H. O. Hoffmann, of Chicago, subject to an annuity to himself, but to be used on his death, preferably to support and develop teaching and research in the care of children.

University of Michigan Medical School.—The Couzens Children's Fund of Michigan, under the direction of the Michigan State Medical Society and the University of Michigan Medical School, will present a conference on diseases of infancy and childhood at the University Hospital, Ann Arbor, Mich., Nov. 26, 1929.

Correspondence

"BLOOD CULTURES AFTER TONSILLECTOMY"

To the Editor:—The article on blood cultures after tonsillectomy by Rubin, Epstein and Werner in your October issue was of considerable interest to us, as the work was similar to experiments which we performed in 1925. Our blood cultures were taken only on patients who had recently had rheumatic fever, with the idea of determining what organism, if any, was forced into the blood stream of such persons following traumatism of one of the apparent foci of infection. We thought that by this means we might possibly obtain the virus of rheumatic fever. It was natural to assume that the virus of rheumatic fever was forced into the blood stream after tonsillectomy; the "lighting up" of an old infection, or, better still, the occurrence of crops of rheumatic nodules immediately following the operation in such cases tended to substantiate this point of view. Moreover, the work of Schottmuller, who demonstrated the presence of bacteremia during curettage in septic abortion; of Siefert who obtained 60 per cent positive cultures during bone operations and 40 per cent positive cultures in operations on the soft parts; and of Libman, who in many papers described postoperative and traumatic bacteremia, gave further evidence for that assumption. Accordingly, blood cultures were taken from suitable patients at the following times: (*a*) one hour before operation, as a control; (*b*) just after removal of one tonsil, or during the course of the removal of the second tonsil and (*c*) two hours after the completion of the tonsillectomy.

We considered as suitable patients those who had a definite history of rheumatic infection; in all there were eleven such cases. The first blood cultures, i. e., the controls were, of course, all negative. The cultures taken two hours after operation were also all negative. However, three of the cultures taken during the operation showed the presence of bacteremia. In one case the organism was a hemolytic streptococcus, which grew in all the mediums; in another case, the organism was an anhemolytic streptococcus which grew only in the anerobic mediums; the third case showed a streptococcus which grew only in one flask and failed to grow on subculture. The culture mediums were the ordinary aerobic and anaerobic culture mediums used in the laboratories of The Mount Sinai Hospital, New York, and were prepared under the direction of Dr. Gregory Shwartzman. Included in these mediums, in addition to the plain broth and dextrose broth flask and agar plates, were tomato extract broth (10 per cent tomato extract, 2 per cent peptone, 1 per cent dextrose and broth): the Rosenow medium (sterile calves brain, marble and 10 cc. plain broth); and the Noguchi medium (sterile rabbit's kidney, 10 per cent peptone, 20 per cent dextrose and ascetic fluid).

HERMAN SCHWARZ, M.D., and
IRVING A. FRISCH, M.D., New York.

Abstracts from Current Literature

Anatomy and Physiology

THE LOCALIZATION OF VEGETATIVE CENTERS IN THE CEREBRAL CORTEX. FRANCISZEK WICHERT, J. f. Psychol. u. Neurol. **37**:693, 1929.

A girl, aged 14, was admitted to the hospital; her previous history, except for measles and pertussis in childhood, was unimportant. On admission, she said that suddenly and without apparent cause her right hand trembled for two minutes; she did not lose consciousness. On the next day, immediately after carrying a heavy barrel, the right hand began to shake again, after which she felt as if "something was running from the tips of the fingers to the throat"; at that time she was unable to speak and felt stiff; she was put to bed but did not lose consciousness; this attack lasted for about five minutes. Similar seizures recurred every three or four weeks, but at the end of six months they also began to involve the left hand, and later both lower extremities, more markedly on the right side. One year after the onset of the illness, the seizures became much more frequent and on several occasions were associated with loss of consciousness and biting of the tongue. At about the same time, the right hand became gradually weaker, and occasionally a series of attacks would be ushered in by minor attacks involving only the fingers of the right hand. Six months after the onset, the patient also noticed that on several occasions the attacks in the right hand would be preceded by cyanosis of the fingers, which began at the tips and was sharply limited at about the middle of the metacarpal bones; the cyanosis disappeared immediately after the attack. At about the same time, she also noticed that the cyanosis was associated with edema of the right hand which also disappeared with the cessation of the seizure. The right hand showed desquamation of the superficial skin and atrophy. The skin of this hand was smooth and shining, and was always warmer and more moist than that of the left. During the seizures the trophic and vasomotor disturbances were sharply separated from the adjacent normal skin. The right hand was thinner from the beginning of the illness. Roentgenologic examination showed the outlines of the bones of the right hand to be very distinct. The tendon reflexes on the right were always hyperactive and after the more severe seizures she would drag the right lower extremity. No pathologic reflexes were ever observed. The cranial nerves were normal except that following a series of attacks there was slight paresis of the right facial and oculomotor nerves which would soon disappear. The Wassermann reaction was negative in the blood and cerebrospinal fluid. Inoculation (intracutaneous and intraperitoneal) of a guinea-pig with cerebrospinal fluid gave negative results. The fundi were normal. Two years after the onset of the illness, the patient died in status epilepticus.

The mental examination revealed normal intelligence, memory, attention and behavior; just before the attacks she would become emotionally unstable and irritable with a tendency to depression and crying.

Microscopic examination showed diffuse changes in the ganglion cells throughout the entire central nervous system. These changes consisted of chromatolysis, disintegration of the intracellular fibrils and protoplasm with eccentric displacement and unusual dark staining of the nuclei. The predominant lesion in the cells was that of a severe degeneration. In the frontal lobe some areas showed acute cell disease (Nissl). In the left occipital lobe the changes in the second and fourth layers resembled those in the third and fifth. Ameboid transformation of the glia cells and an increased number of Hortega cells were in evidence everywhere. Fatty degeneration of the ganglion and glia cells and of the vessels was most evident in the cornu ammonis. Some parts of the cerebellum also showed an increased number of glia cells. Finally, the part of the motor cortex representing the right forearm showed an old scar, 7 by 5 by 0.5 mm., consisting of glial

and connective tissue fibers with an absence of ganglion cells and myelin fibers; in the area adjoining the scar, ganglion cells and myelin fibers were present but markedly diminished in number. There was no sclerosis of Ammon's horn. This scar, the author believes, was the anatomic substratum for the seizures and the diffuse changes in the ganglion and glia cells, the results of the seizures. The fact that this scar, which must have existed several years before the first seizures appeared two years before the examination, did not give rise to any clinical manifestations until that time, the author explains on the theory of changes in the body chemistry from the endocrine disturbances that must have existed at the time of puberty.

The history, clinical course and pathologic observations lead Wichert to believe that the case was one of symptomatic epilepsy following an old encephalitic process, perhaps in the course of the patient's measles or whooping cough. The vasomotor and atrophic disturbances in the involved upper extremity are also attributed to the scar in the region indicated, and it is therefore concluded that special vasomotor centers for individual limbs and segments of limbs must exist in the cerebral cortex, especially in its deeper layers. The proof offered for this hypothesis, which has originally been presented by Monakow, is too speculative and not wholly convincing to the reviewer, especially as the author admits that the entire brain was not studied in serial sections.

KESCHNER, New York.

THE CEREBROSPINAL FLUID, THE ANATOMY OF THE SUBARACHNOID SPACE AND THE BARRIER BETWEEN THE BLOOD AND CEREBROSPINAL FLUID. L. L. PAPADATO, Collected Papers, Neurological Clinic, State Medical Institute, Odessa, U. S. S. R. **19**:99, 1929.

In a prolonged article, the author gives a complete review of the work and literature of Russian and foreign investigators in regard to the whole subject of cerebrospinal fluid. Embryologic and physiologic data point to the conclusion that the choroid plexus has a secretory function. If this is so, then the question of innervation naturally arises. An investigation of this problem consisted in a series of experiments in which the central ends of the cervical sympathetic fibers were stimulated. In the first series of experiments the internal carotid sympathetic plexus was stimulated. In the second series the central end of the vagosympathicus was stimulated, and in the third series the vagus was stimulated after it had been cut and the central nerve fibers had been allowed to degenerate.

In the first series of experiments dogs were anesthetized, and the plexus lying over the carotid artery was exposed. A cistern puncture was done at the same time. The proper location of the plexus was ascertained by stimulation of the sympathetic to see if there was dilatation of the pupil. As soon as a normal rate of flow of the spinal fluid began the experiment was commenced. A small electrode was applied to the plexus. A Dubois-Raymond coil was used with a current discharging from one dry cell. As a result of irritation of the sympathetic nerve fibers, there was a marked increase in flow of the cerebrospinal fluid. The rate of the flow was recorded automatically by a small moving picture camera.

As there is a great deal of uncertainty in locating the plexus and the whole operation is technically difficult, the second series of experiments was undertaken in which the vagus-sympathicus was exposed and the central fibers were stimulated. As in the previous experiment, a cistern puncture was done. The central ends of the sympathetic nerve fibers were stimulated by a very weak faradic current. The rate of flow of the cerebrospinal fluid was measured before the stimulation with the electrical current, during the period of irritation and also afterward. The rate of flow was in the inverse ratio to the length of the experiment. With irritation of the central nerve fibers there was an increase in the rate of flow of the cerebrospinal fluid, but with each succeeding irritation the flow was less and less. Thus in the beginning of the experiment a drop came out about every thirty-seven seconds, while toward the end of the experiment it took on an average two hundred and forty-eight seconds for each drop of fluid to appear.

The author comes to the conclusion that the irritation of the cephalic end of the vagus-sympathicus leads to an increase in the cerebrospinal fluid pressure with increase in the rate of flow. This is due to the sympathetic nerve fibers because when the influence of the sympathetic is eliminated by allowing them to degenerate these phenomena are not observed. The author also concludes that the increase in the cerebrospinal fluid pressure is due in the experiments to the increased secretion of the choroid plexus. The sympathetic nerve fibers are the secretory nerves of the choroid plexus.

KASANIN, Boston.

Pathology, Bacteriology and Biochemistry

THE AMOUNT OF CHOLESTEROL IN THE BLOOD OF HEALTHY AND SICK CHILDREN: ITS BIOLOGIC ACTION. L. VARONE, Riv. di clin. pediat. **27**:599, 1929.

The cholesterol of the blood of twelve normal infants under 1 year of age averaged 1.15 per cent (maximum 1.40 per cent, minimum 0.85 per cent); in nine children, from 2 to 8 years of age, the average was 1.37 per cent (maximum 1.50 per cent, minimum 0.85 per cent). In children with spasmophilia or rickets, the cholesterol of the blood is definitely increased during the acute condition as compared to a normal percentage after the child is cured.

In children with diphtheria, the cholesterol of the blood is diminished.

In tuberculous children, the cholesterol is low when the condition of the patient is grave, but becomes elevated as the nutrition and reaction of the patient to the disease improve.

In several children, cholesterol was given by injection; it was impossible to modify consistently the percentage of cholesterol in the blood by this means.

HIGGINS, Boston.

Nutrition

CHEMICAL CHANGES IN THE BLOOD DURING FASTING AND SUBSEQUENT REFEEDING: II. INORGANIC COMPONENTS. S. MORGULIO and V. L. BOLLMAN, Am. J. Physiol. **84**:350, 1928.

There is little change in the electrolytes of the blood in fasting. The chief changes are a fall in chloride content accompanied by a rise in bicarbonate. This disappears after a few days of refeeding and is not permanent. The calcium shows a large diminution during inanition. This effect, however, is peculiar to the plasma, and may be due to a shift in the globulin-albumin ratio during fasting.

KUGELMASS, New York.

THE NATURE OF THE PROTEIN SURROUNDING THE FAT GLOBULES IN MILK. R. W. TITUS, H. H. SOMMER and E. B. HART, J. Biol. Chem. **76**:237, 1928.

The protein surrounding the fat globules in cow's milk was found to have the same sulphur, phosphorus and tryptophane content as caseinogen, and the same specific rotation ($\alpha \frac{20}{D}$ in N/2 NaOH = 85°). The serum of rabbits immunized to casein gave a precipitin reaction with the fat globule protein, and vice versa. It is concluded that the protein surrounding the fat globules is caseinogen.

KUGELMASS, New York.

A STUDY OF MALNUTRITION IN CHILDREN. H. HARRIS PERLMAN, M. J. & Rec. **130**:148 (Aug. 7) 1929.

Four hundred malnourished children who came under observation at the health center were studied to determine the influence of parental and preschool diseases on malnutrition. From this study, the author drew the following deductions:

1. The season of the year appears to affect nutrition; more cases of malnutrition were found during the summer months.

2. Malnutrition is a relatively frequent condition in childhood.

3. There were comparatively few children who could be considered dangerously undernourished. Moderate malnutrition occurred with comparative frequency, while the slightly undernourished children comprised the largest group.

4. Physical defects bear a direct relation to various degrees of malnutrition.

5. Chronic prenatal diseases do not materially influence the occurrence of malnutrition in offspring. The common infectious diseases of childhood cannot be ignored entirely as predisposing or direct influences on malnutrition. Of these, the commonest was measles.

6. Boys were affected as frequently as girls.

7. The common defects that were found to be associated with malnutrition were dental caries, cervical adenitis, diseased tonsils, secondary anemia, poor posture and defective vision. All of these defects were preventable, and in many cases capable of correction. Focal infection, such as diseased tonsils, is one of the most common causes of malnutrition.

8. To succeed in overcoming malnutrition, one must study the undernourished child; to this end, the cooperation of parents, school authorities and sociologists is needed.

WILLIAMSON, New Orleans.

1. THE VITAMINS OF ORANGE JUICE. 2. THE VITAMINS OF COMMERCIALLY CONCENTRATED ORANGE JUICE. S. G. WILLIMOTT, Biochem. J. **22**:67 and 535, 1928.

1. California navel and Valencia oranges were used. The juice gave a faint positive reaction to the color test with arsenic trichloride or antimony trichloride. Vitamin D was supplied in vitamin A titration by a suspension of 2 per cent irradiated chloresterol in paraffin; 5 cc. contained sufficient vitamin A for normal growth of young rats, and 10 cc. sufficient vitamin B; in both cases the addition greatly increased the animal's intake. On Zucker's diet, 10 cc. of juice failed to alter the fecal p_H in fourteen days; cod liver oil made the feces acid in four days. It is therefore probable that orange juice does not contain vitamin D. It was also observed that wheat starch, but not rice starch, contains traces of vitamin B.

2. The juice was concentrated in vacuo at 37 C. to one-seventh its original volume and was preserved in sterile jars in the cold. For feeding to the rats, it was diluted with distilled water to its origin volume; 5 cc. of the diluted concentrate contained sufficient vitamin A, and 10 cc. sufficient vitamin B, for growth. Hence, no loss of either of these vitamins on concentration and preservation was detected.

KUGELMASS, New York.

1. THE DETERMINATION OF AMINO-ACIDS IN THE BLOOD: A NOTE ON FOLIN'S METHOD. 2. THE AMINO-ACID CONTENT OF THE BLOOD OF CHILDREN IN HEALTH AND DISEASE. S. EDGAR, Biochem. J. **22**:162 and 168, 1928.

1. The figure obtained for amino-acid N in blood filtrates depends on the alkalinity of the solution; it is higher when the solution is made more alkaline; thus, on adding 1 cc. of sodium carbonate, 6.68 mg. per hundred cubic centimeters was found, and the amount rose to a constant maximum of 8.92 mg. per hundred cubic centimeters with 1.7 cc. alkali. The blood of fasting subjects contains slightly less amino-acid N than that of nonfasting persons (8.23 mg., instead of 8.97 mg. per hundred cubic centimeters). The difference is brought out more clearly by studying the distribution instead of the average; 30 per cent of the blood of nonfasting persons lies above 10 mg., and 30 per cent of that of fasting persons between 8 and 9 mg. There is no change in the amino-acid N in rheumatism and chorea and no regular change in nephritis, but there is a decrease in renal and celiac infantilism.

KUGELMASS, New York.

PHOSPHORUS COMPOUND OF MILK: IV. PRESENCE OF ADENINE NUCLEOTIDE IN MILK. H. D. KAY and P. G. MARSHALL, Biochem. J. **22**:416, 1928.

Goat's milk is precipitated with picric acid paste; after the removal of potassium picrate the filtrate is neutralized and filtered, and 20 per cent of uranyl nitrate is added to it. The precipitate is washed with water, dissolved in sulphuric acid and precipitated with mercuric sulphate. This precipitate is decomposed in the usual way, and the filtrate treated with magnesia mixture. The nucleotide is then precipitated as a silver salt, the salt is decomposed by hydrogen sulphide, and the nucleotide finally precipitated with alcohol. It did not analyze for pure adenine nucleotide, but gave pentose reactions after hydrolysis with hydrochloric acid; following hydrolysis with mercuric sulphate, material could be precipitated by ammoniacal silver nitrate, and finally a filtrate was obtained which was apparently adenine picrate. This treatment removed about half the organic phosphorus. Kidney phosphatase removed the whole of it; the original substance contained no free phosphate. About 3.3 mg. of nucleotide was obtained from 100 cc. of milk, equivalent to 0.3 mg. of organic acid-soluble phosphorus out of 13 mg. present.

KUGELMASS, New York.

THE DUAL NATURE OF WATER-SOLUBLE VITAMIN B: II. THE EFFECTS UPON YOUNG RATS OF VITAMIN B2 DEFICIENCY AND A METHOD FOR THE BIOLOGICAL ASSAY OF VITAMIN B2. HARRIETTE CHICK AND MARGARET H. ROSCOE, Biochem. J. **22**:790, 1928.

Rats weighing from 40 to 50 Gm. are placed on the following diet: purified caseinogen, 100 Gm.; rice starch, 300 Gm.; cottonseed oil, 75 Gm.; salts, 25 Gm. and water, 500 cc., together with from 0.05 to 0.1 Gm. of cod liver oil daily; the diet is heated in steam for three hours at 100 C. to prevent rejection. The animals must be kept in cages with false screen bottoms to prevent coprophagy. After two weeks on the diet they are given daily 0.1 cc. (equivalent to 0.6 Gm. of dry yeast) of an antineuritic yeast concentrate, to supply vitamin B1. The caseinogen must be purified by precipitating with acetic acid, washing by decantation with 0.05 per cent acetic acid twice daily for two weeks, extracting four days in Soxhlet's apparatus with from 50 to 70 per cent alcohol, drying and finally heating for three days at 120 C. Unextracted caseinogen contains vitamin B2. Symptoms of B2 deficiency appear in from two to three months; dermatitis and loss of hair of the eyelids, head, abdomen, nose and mouth; sunken eyes; inflammation of the nostrils, ear tips and digits, and hematuria. Death occurs in about fifteen weeks. The animals remain at a constant weight throughout. For the assay of vitamin B2 the source of the vitamin should be given after about four weeks on the basal diet (plus the cod liver oil and yeast concentrate); the response to its administration is immediate growth proportional, within limits, to the amount of vitamin B2 supplied; the same animal can be used for two tests, provided that the smaller dose is given first, or that two weeks on the basal diet elapse between the tests.

KUGELMASS, New York.

A METHOD OF ASSAY OF THE ANTIRACHITIC VITAMIN D. K. H. COWARD, Quart. J. Pharm. **1**:27, 1928.

A particular sample of irradiated ergosterol is adopted as a standard for antirachitic potency; 0.0001 mg. of this preparation, under the condition of the test, will cause complete healing of the induced rickets. For a preliminary period of three or four weeks, young rats (from 50 to 60 Gm. in weight) are fed on a diet rich in calcium and low in phosphorus; this is followed by a test period of ten days, during which the substance under assay is given to them daily. The rats are killed, and the degree of healing is estimated by a macroscopic examination of the ulna and radius by means of the "line" test of McCollum, Simmonds, Shipley and Park. This is described in detail, and a useful illustration given which pictures the degree of healing obtained in an actual experiment from different doses of the

standard preparation. As the severity of the induced rickets and the degree of healing varies from litter to litter, even under identical conditions, a direct comparison is made of the sample under assay with the standard by giving one rat no dose at all, to indicate the degree of rickets in the particular litter, and by giving two or three different doses both of the standard and of the sample to the other members of the same litter. The quantity required for complete healing is estimated in both cases, and the dose required of the sample equated to that required of the standard. The equivalent of 0.0001 mg. of the standard is then calculated, and the result expressed in units.

KUGELMASS, New York.

THE GROWTH-PROMOTING PROPERTIES OF VITAMIN D. A. L. BACHARACH, Quart. J. Pharm. **1**: 49, 1928.

Five litters of rats, comprising six animals each of the same sex, were divided into two series of three groups each. All received the same basal diet (a modification of that described by Drummond and Watson), with the exception that the source of fat was varied. Group A was given refined edible cottonseed oil through which air had been drawn for seventy-two hours at from 110 to 130 C.; group B received hardened soya bean oil and group C the unhardened oil. In addition, one series of rats was given a cod liver oil concentrate as a source of vitamin D. The hardened oil was found by a separate experiment to be devoid of antirachitic effect, but to contain some vitamin A, as shown by the color test. Both the rats on refined cottonseed oil and those on hardened soya bean oil showed slight growth, which was increased by the addition of vitamin D; those on the unhardened oil gave better growth, which was also increased on addition of the antirachitic vitamin. The author concludes that liquid soya bean oil is a good source of vitamin A free from vitamin D, that the process of hardening has a destructive effect on vitamin A and that vitamin D has growth-promoting properties.

KUGELMASS, New York.

ON THE INFLUENCE OF NUCLEIC ACIDS OF VARIOUS ORIGIN UPON THE GROWTH AND LONGEVITY OF THE WHITE MOUSE. T. BRAILSFORD ROBERTSON, Australian J. Exper. Biol. & M. Sc. **5**:47, 1928.

Thymus nuclei or yeast nucleic acid, given to mice in daily doses, produced little effect on the growth until after the seventh week of age, when the normal senescent loss of weight was delayed. Life was prolonged from 13 to 17 per cent. Spleen nucleic acid caused acceleration of growth after 18 weeks of age; after 78 weeks the treated animals lost weight, so that at 92 weeks the curves for normal and treated animals coincided. Duration of life was only increased about 5 per cent, within the experimental error. It is suggested that the effects of nucleic acid are attributable to the sustainment or enhancement of nuclear material in the cell. The nucleocytoplasmatic ratio may thus increase and longevity be the result. If, however, excess growth accompanies the increased provision of nuclear material, the ratio will not rise and, as in the case of spleen nucleic acid, longevity is not increased.

KUGELMASS, New York.

WHY RICKETS DOES NOT EXIST IN MEXICO. TORROELLA, Arch. de méd. d. enf. **32**:262, 1929.

An exhaustive examination of 6,000 infants failed to reveal a single case of rickets; a further report from the obstetric wards of the Liceaga Hospital showed that no cases of dystocia due to rachitic deformity of the pelvis had been encountered.

If syphilis is one of the sources of rachitic manifestations, as advanced by Hutinel, Mexico with its thousands of infected infants would furnish numerous examples. Torroella argues that the complete absence of rickets demonstrates conclusively that inherited syphilis plays neither a major nor a minor rôle in the

genesis of this disease. He differentiates the stigmas of syphilis in early life from the clinical picture of rickets as follows:

1. True lesions of rickets are usually observed after the sixth month, while the dystrophies of heredosyphilis are noted in the early weeks.

2. Heliotherapy is without effect when the lesions are nonrachitic.

3. Specific treatment is followed promptly by amelioration of symptoms in cases of inherited syphilis, while no result is noted in rickets.

Again, if poisons other than bacterial can lay the foundation for the syndrome that is termed rickets, why, the author asks, does not a population largely given over to excesses in intoxicating liquors furnish cases in great number?

Funk declares that rickets is an avitaminosis. This definition may be accepted if light is considered as a vitamin.

The three principal factors in the prevention of rickets among Mexican infants are: (*a*) sunlight, which is rich in ultraviolet rays, especially on the central plateau; (*b*) foodstuffs providing major quantities of calcium, and (*c*) maternal nursing.

The construction of Mexican habitations, with the inevitable patio, is a powerful aid in heliotherapy, while the equability of the climate contributes notably to prophylaxis.

Tables showing the unusual amounts of calcium contained in native dishes are quoted in defense of the thesis that the fixation of this element by the ultraviolet rays constitutes one of the chief reasons for the nonexistence of rickets in this favored territory.

AMESSE, Denver.

THE DIAGNOSTIC VALUE OF THE DETERMINATIONS OF ORGANIC ACIDS AND AMMONIA IN INFANTS' STOOLS. RÉNÉ MATHIEU, Bull. Soc. de pédiat. de Paris **27**:36 (Jan.) 1929.

In acute diarrhea the organic acids in the stools of breast-fed infants are higher than those in the stools of infants fed on formulas of cow's milk. However, the determinations vary a great deal, even in the same infant, and they are of no prognostic value. They do not help in the differentiation between diarrhea due to enteral causes and that due to parenteral causes. Only 10 Gm. of stool was used for each determination, so that the figures obtained bear no relation to the total amount excreted in the stools for twenty-four hours. In subacute and chronic indigestion, the determinations of organic acids were of no value.

The ammonia content of the stools at the onset of acute diarrhea is normal or low, and the urinary phenols are high. During the course of acute diarrhea, the ammonia rises from 4 or 5 cc. to 8 or 10 cc. (tenth-normal sodium hydroxide was used for 10 Gm. of stool). This increase means that there is an exaggerated action of the proteolytic flora of the intestine. The reaction of the stools to litmus is normally alkaline in infants fed on cow's milk, and acid in those fed on breast milk. In abnormal conditions, the reaction varies according to many factors.

The conclusions drawn are:

1. Determinations of the organic acids, ammonia and reaction to litmus of infants' stools are of no value. 2. The frequent elevation of organic acids in the stools in parenteral infections shows that disturbances in the general condition of the infant have a bearing on intestinal function. 3. The relation between infections, alterations in nutrition, and intestinal reactions explains why the study of the stools in infants is not of the same value as that in adults.

BENJAMIN, Montreal, Canada.

WHY RICKETS DOES NOT EXIST IN MEXICO. JOSE J. MARTIN GONZALES, Bull. Soc. de pédiat. de Paris **27**:42 (Jan.) 1929.

The author refers to Marfan's view that rickets is due to chronic toxemia which produces a morbid overactivity of the lymphatic system. Hutinel believes that it is a manifestation of a general nutritional disorder affecting especially

the bones, due to a hereditary predisposition, and particularly present in hereditary syphilis. Bouchard, in 1882, stated his belief that rickets was a nutritional disorder producing excessive growth of the bone tissue with insufficient calcification of that tissue. In Mexico, the author saw no case of rickets in 6,000 patients, and there was not a single patient with a rachitic pelvis in the obstetric clinic. He ascribes the absence of rickets in Mexico: (1) the fact that the amount of sunlight is distributed evenly over every season, especially in the central plateau; (2) the food, which is rich in calcium, and (3) breast feeding.

Marfan points out that it used to be said that there was no rickets in Turkey; yet he saw many cases of rickets in a hospital in Constantinople. Similarly it was thought that Japan and Africa were free from rickets, but cases have been observed in these countries. He believes that syphilis and infections from the last month of intra-uterine life to the end of the first year predispose to the development of rickets. Marfan believes that when syphilis is prevalent and rickets is not prevalent, the situation is due to the food, climate, etc., and he points out that general paresis and tabes dorsalis are rare there, even though they are ordinarily common whenever syphilis is prevalent.

Comby believes that syphilis and infections may predispose to rickets by disturbing the general nutrition, but they have no specific action; there is no syphilitic rickets.

BENJAMIN, Montreal, Canada.

PROTEIN CEREALS AND "ALEURONS." L. RIBADEAU-DUMAS, E. ANDRE, R. MATHIEU and WILLEMIN, Bull. Soc. de pédiat. de Paris **27**:63 (Jan.) 1929.

In 1855, Hartig described an oily grain free from starch, which he called aleuron. Several years later, it was shown that this form of grain had nutritious qualities. It was particularly rich in protein and contained a large amount of mineral salts, such as calcium and magnesium phosphates. The authors fed aleurons to a group of infants who were deprived of milk because of digestive disturbances; all the infants did well. Some were fed on this diet for about three months and showed no bad effects. The authors quote the case of an infant in China who was fed a soy bean mixture. The old objections that vegetable proteins were not properly utilized by the body and that they did not contain the necessary growth-producing factor are not well founded. The authors point out that the feedings contain tryptophan and that they are rich in inorganic salts. Willemin is making further studies on these feedings.

BENJAMIN, Montreal, Canada.

PROPERTIES OF THE CHONDROID ZONE OF RACHITIC BONES. A. POLICARD and M. PEHU, Compt. rend. Soc. de biol. **99**:83, 1928.

The cartilage of the chondroid zone has special physicochemical properties. It is remarkably transparent compared with the opaline appearance of epiphyseal cartilage. It rapidly swells in water; this imbibition fails in faintly acid water, whereas the swelling of connective tissue increases. This property is of great importance in the modification of processes of ossification.

KUGELMASS, New York.

THE THERAPEUTIC USE OF VITAMIN C IN THE INFANT. P. ROHMER, Rev. franç. de pédiat. **5**:293 (June) 1929.

The vitamin C used in this study was a yellow powder extracted from fresh cabbage in the absence of oxygen. It effected a prompt cure in infantile scurvy and gave excellent results in four cases of celiac disease and in five cases of hypothrepsia. This preparation was of advantage because it could be given in large amounts when orange juice produced diarrhea. With its use, the appetite improved and a marked gain in weight resulted. Also, it apparently improved the stools.

LESLIE, Evanston, Ill.

NUTRITIONAL EDEMA. E. A. VÁZQUEZ, Bol. Soc. Cubana de pediat. Havana **1**:415 (July) 1929.

A child, aged 3 years, presented edema of the lower extremities and the abdomen. Nothing was found on examination of the circulatory system; other systems were also normal. The blood showed a slight anemia, and the urine was normal. Chemical examination of the blood showed the cholesterol content to be 210 mg., the other constituents being approximately normal. The child was put on a high protein diet and given 0.0065 Gm. of thryoid substance twice a day. Caffeine was given later. The improvement was prompt. The cholesterol fell to 160 mg. Vázquez believes that this was a true case of nutritional edema, the lipotic ratio of cholesterol over fatty acids being deranged sufficiently to overthrow the albuminoid ratio of the blood serum.

SANFORD, Chicago.

DIGESTIVE AND NUTRITIONAL DISTURBANCES IN THE NURSLING. SALVADOR E. BURGHI, Arch. españ. de pediat. **13**:139 (March) 1929.

The author considers excessive heat one of the chief aggravating factors in diarrheal alimentary disturbance, regardless of whether the disturbance is enteral or is due to parenteral causes. Excessive heat directly favors acute dehydration. The cellular structure of the organism can tolerate dehydration up to a certain point, beyond which the cell loses the ability to fix water; when this point is reached, attempts to hydrate tissue by artificial means will fail.

SCHLUTZ, Minneapolis.

FOOD VALUE OF FATS: III. J. OZAKI, Biochem. Ztschr. **192**:428, 1928.

In decreasing order of food value for rats, the fats examined were butter, rape seed oil, arachis oil, olive oil, liver fat, herring oil, cotton seed oil, triolein, beef fat, lard, whale oil and sardine oil. Liver oil has a high food value, probably according to its vitamin content.

KUGELMASS, New York.

ANTIRACHITIC PRINCIPLE IN GRASS GROWN IN THE DARK. W. VOLTZ and W. KIRSCH, Biochem. Ztschr. **193**:281, 1928.

Grass grown on a synthetic medium in absolute darkness to a height of from 10 to 12 cm. effects healing in cases of rickets; the antirachitic value is somewhat less than that of normal grass, owing to the lower concentration of salts.

KUGELMASS, New York.

ACUTE AND CHRONIC RICKETS. H. VOLLMER, Deutsche med. Wchnschr. **55**:1132 (July 5) 1929.

The author suggests that a classification of rickets into acute and chronic will not confuse the reader's understanding of the disease but will help to bring out certain phases of the disease picture not usually described in textbooks. Even aside from late rickets and osteomalacia, the disease presents a varied picture. He cites illustrative cases: one in which the child is brought to the physician for a sudden change in disposition entirely due to rickets, and another in which the disease is discovered during routine examination for some other condition. The latter type of rickets, which is often overlooked until an osseous deformity brings the child to the orthopedist, is really the more alarming because of its insidious nature.

In order to take proper prophylactic measures in both types, Vollmer finds it necessary to go into the causative factors of rickets. The development of rickets is dependent on two major factors: the rapidity of growth and the amount of ultraviolet light obtainable. Acordingly, he groups the cases seasonally and according to age. In the winter months, when there is little ultraviolet light, the small infant up to 8 months of age and especially the premature infant, in whom

growth is most rapid, will develop acute rickets. The older child and those who have reserve stores of actinic rays will develop chronic rickets. In summer, when there is a great deal of ultraviolet light, the younger infant will develop chronic rickets and the premature infant, because he cannot be exposed to sunlight, will probably still develop acute rickets, while the older child will be comparatively free from the disease. The other natural factors in the etiology are also discussed. The author insists that the line of demarcation cannot be definite and that the cases progress. Furthermore, it is to be understood that this scheme does not deal with the individual and that it is largely based on the seasonal occurrence. While a child may escape the disease, due to a sufficient store of vitamin, this store may become exhausted at the end of the winter and the rickets may develop in the early spring.

As a further argument in favor of the division into two groups, Vollmer cites the phosphate content of the blood. This, again, cannot be an absolute criterion for the division into acute and chronic but, with the clinical and roentgenologic observations, it helps to permit such a division.

SCHERER, Duluth, Minn.

SO-CALLED SUMMER RICKETS: A CRITIQUE OF AN ARTICLE BY SCHOENEN. MAX KLOTZ, Deutsche med. Wchnschr. **55**:1177 (July 12) 1929.

Before entering into his arguments to justify his skepticism of Schoenen's diagnosis of "summer rickets," the author shows that two pediatricians may place an entirely different value on the signs of beginning florid rickets. He says, furthermore, that a thorough investigation by one of his assistants showed that craniotabes in a normal breast-fed infant is not always due to rickets, because the roentgenogram and the serum phosphate may be normal and all other symptoms lacking.

He says that Schoenen makes his diagnosis entirely from the clinical observations, and that roentgenologic and serologic data are desirable for confirmation. Also, a large number of cases reported are of very young infants on breast feeding or mixed feeding in whom only craniotabes is noted. About these the author is especially skeptical, in view of his own observations. He agrees with Schoenen that many cases of rickets appear late in the spring or in the early summer and are due to bad surroundings and poor care and feeding. However, Klotz believes that even these children are affected by the disease only when there is a "constitutional disposition to rickets."

The meteorologic explanation that Schoenen builds up is skilfully employed, but the author's arguments seem to discount it entirely. In a summary the author says: "Because of the lack of radiologic and serologic studies, the report by Schoenen permits the question whether or not the marked increase of rickets in the summer months does not include a number of cases of the so-called physiologic cranio-tabes. The attempt to explain this increase on a meteorologic basis is not acceptable."

SCHERER, Duluth, Minn.

ISO-AGGLUTININS IN BREAST MILK. B. EPSTEIN and E. PODVINEC, Jahrb. f. Kinderh. **121**:123 (Sept.) 1928.

Since the more frequent practice of blood transfusion, more careful studies have been made of isohemagglutination. Epstein and Podvinec studied the relationship between the isohemagglutinins in the blood and in breast milk. They used the drop method microscopically.

The following is a brief summary of their results. Of 100 specimens of breast milk there were no iso-agglutinins in 78; in 21 cases there was no iso-agglutination between milk and blood. In 61 instances, there was demonstrated the same group iso-agglutinin in the breast milk as in the blood. In 28 instances, there were doubtful reactions. In all instances agglutination occurred between such groups as do not usually show this phenomenon, leading the authors to surmise the existence of a hitherto latent agglutinogen.

In 50 specimens of colostrum, there were on two occasions iso-agglutinins of uncertain origin.

The authors think that this method of study may lead to a better understanding of the biologic and pathologic facts on which the digestion of breast milk depends.

ABT, Chicago.

THE INCREASE IN WEIGHT OF HEALTHY CHILDREN DURING THE FIRST YEAR OF LIFE. A. O. KARNITZKY, Jahrb. f. Kinderh. **121**:277 (Oct.) 1928.

On the basis of a study of sixty-five breast-fed children during the first year of life, Karnitzky presents the following facts:

A healthy and normal infant tends constantly to an increase in weight according to its constitution. This is manifest in the daily increase in weight. In the majority of children, the greatest daily gain (from 35 to 45 Gm.) occurs during the first month of life.

According to body weight, there are two large groups of children: small, average and above the average in weight. In each group, there are children with slight and with marked powers of growth. One group may show marked growth in the first three or four months, others in the following three to six months. There are three marked influences on the tempo of growth: (1) the constitution, (2) the character of the digestive apparatus and (3) the nature of the child's nourishment. Infections and injuries of various kinds hinder the normal course of development. The first year shows the greatest fluctuations in the weight of the child.

ABT, Chicago.

CALCIUM-SOAP STOOLS. ALICE OELSNER and K. KLINKE, Jahrb. f. Kinderh. **122**:58 (Nov.) 1928.

Oelsner and Klinke note in their chemical investigations that the insoluble calcium soaps of the higher fatty acids of cow's milk may be changed to soluble sodium soaps by sodium carbonate or bicarbonate. Acids liberate fatty acids from the calcium soaps of cow's milk. Keller's alkaline malt soap causes a colloidal state of the calcium soaps. Bile also completely dissolves the calcium soaps.

Oelsner and Klinke note that in the absorption of fat two processes occur: the formation of the salts of the cholic acids with which the earthy alkali soaps may also combine, and the formation of alkali soaps under the action of the alkaline intestinal juices. Under normal conditions, the bile plays an important rôle in the splitting of fats. The improvement in the resorption of calcium through the administration of alkalis depends on an improvement in the relationship between the unsaponified fatty acids and the calcium salts. The removal of part of the fatty acids in the form of alkali soaps improves the calcium and fat relationship with regard to the absorption of calcium. The importance of bile in the resorption of calcium soaps indicates that an insufficient bile secretion is the cause of calcium soap stools.

ABT, Chicago.

NUTRITION OF DIABETIC CHILDREN. H. HIRSCH-KAUFFMANN and E. SCHÄDRICH, Jahrb. f. Kinderh. **122**:74 (Nov.) 1928.

In their study of children with diabetes Hirsch-Kauffmann and Schädrich first review the various dietary theories and methods of the preinsulin era, emphasizing the tendency to acidosis and the interference with normal development.

All authors are agreed that since the introduction of insulin, children with diabetes may attain normal physical development. Joslin, however, has observed a retardation in growth even under insulin. At any rate, the discovery of insulin is considered a turning point in the fate of the child with diabetes. Yet the authors are of the opinion that not every diabetic child need be treated with insulin as, for example, not every cardiac patient requires digitalis therapy. Children are

susceptible to hypoglycemia and should receive insulin only in the event of a persistent glycosuria on the most suitable diet. Boyd observes that 25 per cent of all children with diabetes may be treated without insulin.

A combination of dietetic and insulin therapy is the method of choice when indicated. Not more than three injections of insulin should be given daily, and the administration of carbohydrates should be so timed as to prevent hypoglycemia one-half hour after the injection of insulin. One unit of insulin presumably takes care of from 1.5 to 2 Gm. of dextrose. However, Freise, Heimann-Frosien and Hirsch-Kauffmann have shown that this is not a constant equivalent. The authors discuss the attempts to produce rigid, exact dietary formulas with precise relationships between the ketogenic and antiketogenic substances. The authors do not follow these rigid schemes but attempt an individual type of dietary. They relate an instance in which the child had a glycosuria of 10 Gm., and after increase of the carbohydrate from 112 to 164, without any other changes, the glycosuria disappeared. They decided that the insulin apparatus of the diabetic patient may respond to overloading with carbohydrate by increased endogenous production of insulin. Since then, they have given generous quantities of carbohydrate with good results in growth and development.

The authors conclude that it is not necessary to adhere to such rigid and stringent dietaries as was formerly thought. As a result of their liberal mixed dietary, they note the absence of acetonuria, improved appetite, normal physical development and the requirement of a lesser amount of insulin.

ABT, Chicago.

THE INFLUENCE OF NUTRITION ON THE COMPOSITION OF FAT TISSUE. KARL MOSSE, Jahrb. f. Kinderh. **122**:151 (Dec.) 1928.

According to Mosse, the qualitative peculiarities of pasty fat tissue are practically unknown. It seems more rigid, is readily movable over the underlying structures and only roughly adapts itself to the muscular motions. It gives the impression of lifelessness. This pasty fat tissue, when removed at operation, retains its original form for many hours in contrast to normal fat tissue. The iodine number and the melting point show no difference between pasty and normal fat. The pathologic changes, therefore, do not reside in the fat itself.

To clarify the question as to what influence a one-sided diet has on the composition of the fat tissue, two pigs were fed on protein, carbohydrate and fat diets. The iodine number, the saponification number, the refraction and the unsaponifiable part were alike in all three groups. However, in the fat tissue of animals fed on protein, the water, ash and protein nitrogen was larger in amount and the fat tissue less than in the controls. Also, the phosphorus was present in greater amount than in the other animals. The correlation of the foodstuffs exerts an influence on the composition of fat tissue. The differences affect not the fat itself but the supporting tissues. It is also possible that the qualities of the pasty fat are dependent on the peculiarities of the supporting tissues.

ABT, Chicago.

FAT METABOLISM IN THE PERIPHERAL TISSUE. W. RAAB, Wien. Arch. f. inn. Med. **17**:439 (June) 1929.

In normal persons, the amount of neutral fat found in capillary and venous blood shows variable differences. The administration of carbohydrate is antagonistic to the retention of fat and favors the passage of fat from the tissues to the blood stream. Insulin has the reverse effect. It prevents mobilization of fat and favors the retention of fat in the tissues. Epinephrine has a similar effect, although it is less pronounced than that of insulin.

Pituitary causes a lowering of the blood fat level but has no specific effect on the mobilization of fat.

The patient with diabetes may show increased mobilization of fat.

In cerebral or hypophyseal adiposity, no specific peculiarities of the blood fat level seem to exist.

In diabetic hyperlipemia, chiefly the neutral fat is increased. The free cholesterol is generally only slightly increased.

SCHLUTZ, Minneapolis.

THE EFFECT OF IRRADIATED SUBSTANCES, ESPECIALLY FATS. OSCAR RIED, Wien. klin. Wchnschr. **42**:1105 (Aug. 22) 1929.

In this preliminary communication, the author claims to have conferred photoactivity on fatty substances by irradiation. He is not able to explain all the results on the basis of the Russell effect, and feels that in some cases at least, actual secondary radiations are given off for a considerable time after the irradiation; in other cases, he asserts that active material particles are given off in definite directions.

The author thus seems prepared for any eventuality.

The experimental details will be published later. Meanwhile, a concern in Vienna is introducing to the trade an ointment produced according to the principles given.

CLAUSEN, Rochester, N. Y.

THE DISTURBANCES OF THE METABOLISM OF PHOSPHATE IN RICKETS: II. THE EXCRETION OF ENDOGENOUS PHOSPHATE. WALTER HEYMANN, Ztschr. f. Kinderh. **46**:575 (Dec. 15) 1928.

The excretion of endogenous phosphate in rachitic and nonrachitic infants was studied. They were fed for a short time on Ringer's solution plus 10 per cent malt sugar, 5 per cent brown sugar and 2 per cent flour and 2 per cent butter cooked together, whereupon the inorganic phosphorus in the serum sank sometimes to unmeasurable values. Nevertheless, there was twice as much phosphorus excreted in the urine in the rachitic infants, even per kilogram of body weight as in the nonrachitic, while the amount of phosphorus excreted in the stool was the same. The hypophosphatemia ran parallel to the content of inorganic phosphorus in the spinal fluid, in vesicles caused by cantharides, in tears and in saliva, which led to the conclusion that even the juices of the rachitic tissue were impoverished as to inorganic phosphorus. It was therefore plausible that while the loss of endogenous phosphates was greater, nevertheless it was due to organic phosphates which are more strongly hydrolyzed in rickets. The pathogenetic meaning of this disturbance of ferment was discussed, and the results of the researches on endogenous phosphate metabolism were used for a closer analysis of the researches on the metabolism by other authors.

EMERSON, Boston.

DISTURBANCES IN THE METABOLISM OF PHOSPHATE IN RICKETS: III. PHOSPHATEMIC CURVES FOR RACHITIC AND NONRACHITIC INFANTS AFTER PARENTERAL INTRODUCTION OF ORGANIC AND INORGANIC PHOSPHATE. WALTER HEYMANN, Ztschr. f. Kinderh. **46**:584 (Dec. 15) 1928.

The phosphatemic curves for rachitic and nonrachitic infants, after parenteral introduction of organic and inorganic phosphate, gave the same differences that Warkany described a short time ago after the oral administration of disodium acid phosphate. They were flatter in rickets, even after the exclusion of different factors of absorption. No difference was shown in the phosphatemic reaction to organic phosphate and to disodium acid phosphate.

EMERSON, Boston.

THE PHOSPHATEMIC CURVE OF NORMAL AND RACHITIC ORGANISMS. J. WARKANY, Ztschr. f. Kinderh. **46**:716 (Dec. 15) 1928.

In the studies reported by Warkany, an increase in the content of vitamin D in rabbits resulted in a rise in the phosphatemic curve. This can be looked on as a sensitive, practical indicator, requiring about twenty-four hours for its production and no special preliminary treatment of the rabbits.

EMERSON, Boston.

Prenatal Conditions

MICROCEPHALY IN TWINS. E. A. WHITNEY. M. J. & Rec. **130**:158 (Aug. 7) 1929.

The author gives a chart which records the cranial measurements in four instances of microcephalic twins. Familial microcephaly is common; from one to seven cases in one family have been recorded. Mentally, microcephalic persons range from the profound idiot to the low grade feebleminded subject. The vitality in the lower grade is quite low; only those of higher mentality survive beyond puberty.

WILLIAMSON, New Orleans.

ARACHNODACTYLIA. MAURICE L. YOUNG, Arch. Dis. Childhood **4**:190 (Aug.) 1929.

The literature is reviewed, and a case is presented. Arachnodactylia is considered to begin in the early months of intra-uterine life. It is a true partial gigantism. The suggestion is made that the disease is closely allied to amyotonia congenita.

PETERMAN, Milwaukee.

HEREDITARY ECTODERMAL DYSPLASIA. JEAN SMITH, Arch. Dis. Childhood **4**:215 (Aug.) 1929.

A case of hereditary ectodermal dysplasia is reported in a child, aged 5 years and 9 months. The fifteen cases reported in the literature are reviewed. No new facts are added.

PETERMAN, Milwaukee.

THORACOPAGI. E. HERRMANN, Wien. klin. Wchnschr. **42**:1016 (Aug. 1) 1929.

A case of thoracopagi is reported, with a discussion of the obstetric aspects and the literature.

CLAUSEN, Rochester, N. Y.

CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEAL ANASTOMOSES. KAMIL JEDLICKA, Časop. lék česk. **65**:1226 (Aug. 13) 1926.

The author gives detailed postmortem observations with photographs of the specimens in three cases. Esophageal anomalies are classed as (1) simple obliteration or partial stenosis, (2) atresia associated with tracheal anastomosis and (3) a normal or slightly stenosed esophagus with tracheal anastomosis. A detailed discussion of the mechanics and theories includes inflammatory obliteration, monopoly of available tissue by rapidly growing lungs, persistence of embryonic occlusion, inflammatory adhesion and traction of differences in growth.

STULIK, Chicago.

ECTOPIA CORDIS PECTORALIS. VACLAV JEDLICKA, Časop. lék. česk. **65**:1913 (Dec. 10) and 1959 (Dec. 17) 1926.

The author gives minute descriptions of the postmortem observations of three cases in new-born infants and one in a guinea-pig. These three cases were found in a series of 49,280 autopsies. In two cases the pericardium was absent, while in the third the pericardial sac was subcutaneous. Two of the children lived for eight days. The sternum and xiphoid process were absent or bifurcated.

STULIK, Chicago.

ECTOPIA CORDIS PECTORALIS. J. PRUSKA and JOSEPH SAIDL, Časop. lék. česk. **65**:1962 (Dec. 17) 1926.

The authors report twenty-five cases in the literature to date. Plethysmographic curves of the heart were made, and marked changes in rhythm and volume were shown on exertion or on crying. Cyanosis was prominent. Moving pictures were made of the exposed heart in action, and again when it was covered with a glass jar to prevent desiccation. The temperature was subnormal throughout, except for the antemortem fever.

STULIK, Chicago.

ECTOPIA CORDIS PECTORALIS; ELECTROCARDIOGRAPHIC STUDY. B. PRUSIK and K. WEBER, Časop. lék. česk. **65**:2003 (Dec. 24) 1926.

Using Edelmann's electrocardiograph and a resistance of 35,000 to 110,000 ohms to increase the capacity of the strings, the authors studied the stormy heart cycle of the ectopic heart. The P-R interval was found to be from 0.13 to 0.15 of the normal relation. The heart rhythm was regarded as normal. Several of the electrocardiograms are included in the paper.

STULIK, Chicago.

Diseases of the New-Born

SCLEREMA NEONATORUM. JACOB SHEER, Arch. Dermat. & Syph. **19**:732 (May) 1929.

The author reports a case of sclerema neonatorum in which cure was presumably obtained by roentgen therapy. Various theories as to the etiology and methods of treatment which have been advocated are mentioned.

SHIRLEY, Iowa City.

STUDIES ON THE LIVING BLOOD CELLS OF THE NEW-BORN. CLAUDE E. FORKNER, Bull. Johns Hopkins Hosp. **45**:75 (Aug.) 1929.

Forkner applied the supravital method of studying living blood cells to fifteen new-born infants and followed the changes for eleven days. He summarized his work as follows:

Polymorphonuclear neutrophilic leukocytes decreased in percentage and absolute values throughout the period of observations, the most precipitous drop occurring during the first days after birth. This behavior was in contrast to that of the lymphoid cells. The latter gradually increased in percentage from the day of birth to the eleventh day. The absolute numbers decreased somewhat during the first three or four days, after which they steadily increased.

Polymorphonuclear eosinophils remained constant, with a slight increase in absolute numbers from the third to the fifth day. Corresponding to this increase, there was a definite rise in percentage values.

The monocytes were high in absolute numbers throughout the first eleven days. They did not participate in the initial decrease of total white blood cells. The monocytes followed neither the lymphoid nor the myeloid elements in their fluctuations.

Polymorphonuclear basophils maintained a fairly constant percentage (0.5 per cent) throughout the period of observations, but their total number was highest on the day of birth.

Neutrophilic myelocytes and reticulated red blood corpuscles were both definitely elevated on the first day and then decreased rapidly.

The total number of red blood corpuscles decreased until about the fourth day, with a slight rise at the end of the first week, and then decreased again on the tenth day. This was in contrast to the hemoglobin values which increased about the fourth day, dropped rather rapidly until the seventh day and then subsided more gradually until the eleventh day.

The number of blood platelets was greater on the day of birth than at any other time, but remained rather constant.

The small number of cases studied does not permit an accurate statistical analysis.

LYTTLE, New York.

RESEARCHES ON THE BLOOD IN MELENA NEONATORUM. MAX GROB, Ztschr. f. Kinderh. **46**:748 (Dec. 15) 1928.

In the Infants' Home in the canton of Zurich, from October, 1916, to December, 1927, fifty patients with melena were studied as to coagulation time, bleeding time and morphologic conditions. In a majority of the cases the birth was normal;

for this reason, it appeared questionable whether severe births and their results come into consideration as a cause of melena. In most cases (98 per cent) the hemorrhages occurred within the first four days and lasted from two to four days. The coagulation time was determined in thirty-nine infants by the Burker method; in normal newly born infants it was from five to five and one-half minutes; in twenty-seven infants with melena, between five and three-quarters and fifteen minutes, and in four infants, from thirty-two to one hundred and fifty-two minutes; that is, increased coagulation time in more than 80 per cent of the cases, only four cases showing normal values. Since infants with melena showed no prolonged coagulation time before the occurrence of hemorrhages, this could not be considered as a disposing agent. At the time of the hemorrhage, in almost all cases, an increase of coagulation time was established. The hemorrhage could be ascribed to this increase but not to the absolute height of the coagulation time or to the prolonged coagulation existing beforehand. The coagulation time diminished and reached normal values with more or less marked variation. This diminution could be looked on as a reaction to the hemorrhage.

In sixteen of thirty-nine cases it was possible to establish a ratio between maximum coagulation time and prognosis, in the sense that corresponding to the severity of the case the coagulation time increased. In twenty-three cases, on the other hand, the severity of the cases varied arbitrarily with a constant increase in coagulation time. As not once in a half of the cases was a ratio between maximum coagulation time and prognosis to be established, coagulation time was not to be depended on to distinguish between mild and severe cases of melena. The treatment with citrated blood and serum, administered subcutaneously or intramuscularly, led to success in most cases, the mortality being only 6 per cent; but a direct influencing of the coagulation time by the treatment could not be established. Coagulation time was influenced by a marked simultaneous occurrence of icterus neonatorum either not at all or only insignificantly. The bleeding time studied in thirty-nine cases was regularly from one to five minutes. It could not be said that there was a prolongation in the newly born, and a disposition to hemorrhage from this cause was not considered. The thrombocytes were studied in forty-two cases by Fonio's method; and three fourths of all cases showed, at the time of the hemorrhage, platelet counts, less than 200,000 per cubic millimeter; that is, a real diminution of the number, not only in comparison with adults but also in contrast to the normal newly born infant of the same age, these usually showing values over 200,000 in the first week of life. The diminution of platelets was, however, not so high grade that it was to be considered the exciting agent of the hemorrhage, which could, however, be so favored. As a result of great hemorrhage, the number of red blood corpuscles sank to 3,500,000 on the average, and on the third and fourth days of the hemorrhage the hemoglobin diminished to 68 per cent. Unripe red cells could be looked on as a normal observation for the newly born period or as an expression of a reaction of the bone-marrow to the loss of blood. The leukocyte count in melena was increased (from 10,000 to 12,000) as compared with adults, but it did not vary from the observations in normal newly born infants, and an infectious etiology could not be derived for it. The differential count gave, in harmony with normal newly born babies, a preponderance of neutrophils over lymphocytes in the first week. Monocytes, eosinophils and basophils showed no abnormal values. The occurrence of myelocytes and other immature white cells, especially in the second week, were looked on as a reaction of the bone-marrow to the loss of blood. The Rumpel Leed phenomenon and the Löwis reaction were always negative. It happened also that in melena, in harmony with the observations of Schloss and Comiskey, certain pathologic changes in the blood could be demonstrated, but of these changes only the abnormal coagulation time could be considered as the eventual cause of the severe intestinal hemorrhage. Whether this really was the only cause of melena could not be demonstrated. It is conceivable that the changes in the intestinal mucous membrane on the one hand, and the changes of the blood on the other, represent coordinated disturbances. What the nature of the toxin was, whether bacterial or protein decomposition products which acted harmfully, there was no way of telling.

EMERSON, Boston.

Acute Infections

FULMINATING MENINGOCOCCUS SEPTICEMIA WITHOUT MENINGITIS. RICHARD MIDDLETON and WILLIAM DUANE, Am. J. M. Sc. **177**:648 (May) 1929.

There are two modes of transmission of the meningococcus to the central nervous system: lymphatic and hematogenous; the former has been considered the more frequent, but recently positive blood cultures have often been found before localization of the infection. For this reason vigorous intravenous therapy has been advocated. In some cases the infection does not reach the central nervous system, being only a septicemia; the condition should then be called a meningococcus infection.

There are two forms of septic meningococcus infection: (1) a prolonged febrile state characterized by arthritic symptoms and a mild roseolar eruption; (2) fulminating cases with widespread purpuric blotches. In these cases death may occur very rapidly. A case of the latter type is reported in a man, aged 18, who died fifteen and one-half hours after the onset of the disease. The spinal fluid was comparatively normal, but the blood count showed 88,550 white cells, many of the cells showing large numbers of intracellular diplococci. A blood culture yielded a virulent strain of meningococci. At autopsy, petechiae were found in nearly all organs, particularly in the suprarenals, which were almost completely destroyed by massive hemorrhages. A sparse growth of meningococci in pure culture was obtained from the surface of the brain.

The authors conclude that meningococcic meningitis is usually preceded by a meningococcus septicemia. They think that vigorous intravenous therapy should always be instituted in case of meningococcus infection.

WAGGONER, Ann Arbor, Mich.

RELATION OF VARICELLA TO HERPES ZOSTER. I. STATISTICAL OBSERVATIONS. T. M. RIVERS and L. A. ELDRIDGE, JR., J. Exper. Med. **49**:899 (June) 1929. II. CLINICAL AND EXPERIMENTAL OBSERVATIONS. Ibid. p. 907.

With regard to the incidence of herpes zoster and varicella at different ages, statistics showed that the former was mostly observed beyond the age of 10 and the latter before that age. While varicella showed a constant seasonal variation, herpes zoster did not do so.

In considering experimental and clinical observations regarding the identity of the two viruses in question the writers concluded that the etiologic factors were not identical in the majority of cases.

RATTNER, Chicago.

THE BACTERIOLOGIC DIAGNOSIS OF WHOOPING COUGH. H. SUGARE and J. W. MCLEOD, Lancet **2**:166 (July 27) 1929.

The authors make a plea for the more widespread use of the bacteriologic diagnosis of whooping cough in cases in which the clinical diagnosis is doubtful. The Bordet-Gengou bacillus was successfully isolated in twelve of twenty-one cases of whooping cough and in all of five cases in which examination was made within the first week of the disease. They list the various points of difference between the Bordet-Gengou bacillus and *Bacillus influenzae*, stating that differentiation between these two organisms is not as difficult as many would have one believe.

LANGMANN, New York.

HERPES ZOSTER AND VARICELLA. DONALD STEWART, M. J. Australia **1**:745 (June 1) 1929.

A boy aged 4 years and 6 months had herpes zoster involving the area of the third right dorsal nerve. He was comfortable and afebrile; the results of physical examination were otherwise negative. Four days later, he developed fever, sore throat, and on the chest and back there was a definite varicella rash with about thirty scattered vesicles. The child and his sisters were said to have had chickenpox three years previously. There is a brief review of the literature.

RATTNER, Chicago.

AN EPIDEMIC OF INTESTINAL INFECTIONS WITH BACTEREMIA AND TOXEMIA IN INFANTS LESS THAN TWO YEARS OF AGE DURING THE SUMMER OF 1927 AT WARSAW. H. BROKMAN and J. KOLAGO, Rev. franç. de pédiat. **5**:315 (June) 1929.

The thirty-five cases discussed, although showing diarrhea, vomiting, fever and severe dehydration, differed from the picture described under alimentary intoxication in that there was no loss of consciousness, the breathing was not of the deep, pauseless type and the withholding of food had no beneficial effect. They differed from colitis in that the general symptoms were out of all proportion to the local manifestations, and no severe lesions of the intestine were found at autopsy. All but one of the thirty-five patients died.

Blood cultures showed a variety of organisms: paratyphoid A and C, paracoli and *Bacillus dysenteriae* Flexner. With such a variety of organisms, it is difficult to understand the epidemiology. The authors hope to get results by the use of vaccine in the event of another epidemic.

LESLIE, Evanston, Ill.

RESULTS OF ACTIVE IMMUNIZATION AGAINST DIPHTHERIA IN BERLIN. E. SELIGMANN, Deutsche med. Wchnschr. **55**:1117 (July 5) 1929.

At the end of 1926, a definite increase in the morbidity of diphtheria in Berlin and also a marked rise in the severity and mortality were noted. This increase continued in 1927 and, as a result, a campaign of active immunization was instituted in Berlin. This article is an analysis of the results after one year, taken from the report of the central health department.

The work was begun in November, 1927. The first steps consisted of the appointment by the central health office of numerous stations in the subdivisions and of giving instruction in the technic. The established infant welfare centers and hospitals in children's work were used as stations for younger children, and the school children were immunized in the schools. Private physicians were urged to immunize their patients and to report to the central department. Elaborate reports were required from the stations. For purposes of record, two products were used.

Reports of the immunization of 88,172 children of all ages were recorded and filed, and it is estimated that about 15,000 more were immunized of whom the central station had no report. No ill effect of any kind is recorded. The children were given one and two injections only.

The results of the carefully analyzed figures show that about one-third as many of the immunized persons became ill as of the remaining population, that the disease was relatively much milder in the immunized and that the mortality rate was 8.8 per cent as compared with a mortality of 9.3 per cent for the total number of patients, immunized and nonimmunized. The report states that of 32,221 immunized persons subjected to the Schick test, 71 per cent gave negative results.

In conclusion, the author states that the immunizing substances are satisfactory but can probably be brought to a higher efficiency, that three injections should be given and that the use is justified by his analysis of the reports.

SCHERER, Duluth, Minn.

CAN THE QUARANTINE REGULATIONS FOR DIPHTHERIA AND SCARLET FEVER BE MADE LESS STRINGENT THAN THOSE FOR MEASLES AND PERTUSSIS? MAX KLOTZ, Deutsche med. Wchnschr. **55**:1228 (July 19) 1929.

The author takes issue with Ascher who suggested that the quarantine restrictions for diphtheria and scarlet fever be made less stringent. The tables given by Ascher are cited. These tables show that while patients with measles and pertussis have not been strenuously quarantined, the morbidity for these conditions has shown a distinct drop for five year periods from 1901 to 1925, as compared with that of the other two diseases. In analyzing these tables, the author shows that the percentage of decrease for diphtheria and scarlet fever is definitely greater

than the percentage of decrease for the other two. Because of the greater fatality of the first two diseases, his opinion is that the regulations cannot be changed without harm.

SCHERER, Duluth, Minn.

THE EARLY DIAGNOSIS OF PERTUSSIS. PHILLIP LEITNER, Jahrb. f. Kinderh. **121**:161 (Sept.) 1928.

Leitner has made thorough and frequent hematologic studies of 200 patients with pertussis. He gives his results as follows:

At a very early stage there is a hyperleukocytosis, the average figures being from 20,000 to 35,000. The maximum value in an uncomplicated case in the convulsive stage varied from 60,000 to 84,000. After six or eight weeks, the count returns to normal. Infants and older children react to pertussis in a like fashion as far as leukocytosis is concerned. In the presence of complications, there is a much higher leukocyte count. (The highest count was 114,500 in an infant, aged 15 months, suffering from pertussis and bronchopneumonia.)

It is an unfavorable prognostic omen when the leukocyte count is lower than is expected in the presence of complications.

There was a constant increase of the lymphocytes at the expense of the polymorphonuclears. The relation of polymorphonuclears and lymphocytes, which normally is 1:1, is 1:2 or even 1:4 in pertussis.

ABT, Chicago.

Chronic Infections

SYPHILIS AND PREGNANCY. S. A. GAMMELTOFT, Am. J. Syph. **13**:194 (April) 1929.

The author discusses the problem of syphilis and pregnancy in Denmark, and draws his conclusions from 1,290 syphilitic women delivered at the Lying-In Hospital of the University Clinic of Copenhagen from 1912 to 1926, the total number of births being 23,383, and the percentage of syphilitic mothers, 5.5. He divides the subject into three classifications: (1) the probability of the transmission of syphilis from the mother to the fetus; (2) the prevention of this transmission, and (3) the precautions by the state and society to prevent the spread of congenital syphilis.

A large percentage of infants born of untreated syphilitic mothers are syphilitic; 194 of 201 such infants were syphilitic. Among the patients treated before pregnancy, either with arsphenamine or mercury, the incidence of syphilis in the offspring was slightly diminished. In those patients treated with arsphenamine before and during pregnancy, the incidence of syphilis was low in the offspring. The author suggests strenuous courses of arsphenamine and mercury or bismuth during the course of pregnancy, this treatment to be instituted as early in pregnancy as possible and carried on as intensively as possible, marked albuminuria being a contraindication for the treatment. He advocates the treatment of all syphilitic mothers, that is, all women who have been known to have been syphilitic at one time, whether or not there is any evidence of syphilis. A woman with a negative Wassermann reaction, who has previously been treated may, during the course of pregnancy, infect the infant and again show evidence of active lesions. In the case of the child, the author recommends that it be treated as soon as it is proved to be syphilitic. If both mother and child have definite evidence of syphilis, or if the child has definite evidence of syphilis, it should be treated. If, however, the evidence of syphilis is indefinite, i.e., a weakly positive Wassermann reaction, the infant should not be treated immediately but should be kept under observation until a definite diagnosis may be established, since a certain percentage of these cases will later prove to be nonsyphilitic.

In Denmark, the law provides that a person may be prosecuted who indulges in sexual intercourse when he knows or suspects himself to be infected with a venereal disease. It is also compulsory to submit to treatment, which is offered free. If the patient refuses to continue treatment, he may be incarcerated for

such time as is necessary to conclude the treatment. The law forbids a syphilitic infant to be suckled by any one except its mother, and holds a wet nurse liable to punishment who nurses another's infant when she knows or suspects that she is syphilitic.

The children who are diagnosed as syphilitic may be placed in institutions for four or five years for treatment, after which they are put out in homes. Likewise, children may be held in these institutions for diagnosis in case there is a question of their being syphilitic.

McBRIDE, Sioux City, Iowa.

A STUDY OF PULMONARY TUBERCULOSIS IN CHILDREN. A. LEVINSON, Am. Rev. Tuberc. **19**:499 (May) 1929.

In 119 cases of active tuberculosis, mainly in negro children, pulmonary involvement was present in 99 instances. Of these 99 cases, 69 patients had miliary tuberculosis, 15 acute tuberculous pneumonia and 15 subacute or chronic pulmonary tuberculosis. Tuberculin tests gave negative results in all cases of miliary tuberculosis. In some in which a positive result was obtained on admission, the reaction became negative after the onset of a miliary process. The pulmonary involvement in both acute and subacute forms was fairly extensive and was mainly caseoulcerative. The prognosis in all of the acute cases was bad. Eleven of the fifteen patients with chronic cases died; one is living with a progressive process; one is doing well, and two have disappeared from observation. The high mortality rate in these cases is attributed to the fact that the disease was far advanced when the patients went to the hospital.

SMITH, Ogden, Utah.

TUBERCULOUS CERVICAL ADENITIS. RICHARD H. MILLER and WILLIAM M. SHEDDEN, Am. Rev. Tuberc. **19**:511 (May) 1929.

Tuberculous lymph nodes of the neck constitute a focus of disease which, early in its existence, may set free tubercle bacilli that are carried about the body, resulting in a mild bacteremia. The bacilli may also extend from the originally infected nodes and invade the contiguous ones, finally involving most of those on one side of the neck. All measures which build up bodily resistance are useful in the treatment for this condition. Ultraviolet light does not seem to have much effect on the lymph nodes themselves, but is valuable for its general effect and for its influence on the healing of sinuses. The roentgen ray is an important method of treatment. Tuberculin is of doubtful effectiveness, but seems to help in some cases. An important fact which must be considered in any form of treatment is that the nodes contain living tubercle bacilli, which are a potential source of a general infection. The authors believe that in a considerable number of cases of early tuberculous cervical adenitis, whether in children or in adults, and often in cases of longer duration in which operation is technically feasible, radical surgical operation is the method of choice.

SMITH, Ogden, Utah.

AN INQUIRY CONCERNING THE RÔLE OF ALLERGY, IMMUNITY AND OTHER FACTORS OF IMPORTANCE IN THE PATHOGENESIS OF HUMAN TUBERCULOSIS. ARNOLD RICE RICH and HOWARD A. MCCORDOCK, Bull. Johns Hopkins Hosp. **44**:273 (May) 1929.

This article, of 100 pages, discusses the whole field in great detail. The section here abstracted is that which discusses "the principles involved in the differences between the effects of tuberculous infection in the adult as contrasted with the child."

The chief differences between tuberculous children and adults found at autopsy are stated as: (*a*) a tendency to a more extensive involvement of lymph nodes in children, (*b*) a greater tendency to fibrous encapsulation and to healing lesions in adults and (*c*) a greater tendency in children to the rapid spreading of lesions and to the development of miliary tuberculosis.

The authors then attempt to show to what these tendencies are due. They discuss the explanation offered by Ranke and Kraus and show why they disagree with both these investigators. They do not believe that the facts warrant the statement that the infected child has an acquired resistance greater than that of the infected adult. They do not consider that the intensity of the tuberculin reaction is an indication of the degree of resistance. They do not believe that infants and young children are "deficient in their ability to react fibrously to irritation and injuries of all kinds," and give a number of well known examples of rapid healing and formation of scar tissue in children. They do not consider that the formation of connective tissue is a factor in checking the progress of the tuberculous lesion, but is rather the result of resistance. In place of Krause's aphorism that "the patient is as resistant as the shell of his tubercles," they believe that the patient is as resistant as his acquired ability to hold in check the growth of tubercle bacilli.

The authors quote the well known fact that aboriginal adults coming in contact with tuberculosis for the first time develop the "childhood type" of infection, thus showing that it is no inherent difference between the body of the adult and that of the child which causes the difference in the reaction of the two age groups to the infection.

Ranke's division of tuberculous infection into three stages somewhat analogous to those of syphilis — first the stage of primary infection, second the stage of hypersensitiveness and low resistance (childhood tuberculosis) and third, the stage of high resistance (adult tuberculosis), is discussed, and the authors show why they consider this division artificial and unsupported by fact.

The importance of the number and virulence of the organisms entering the body during the first infection is emphasized. A large number of virulent organisms entering a body before resistance has been developed continue to live and propagate; allergy with its necrotizing influence develops, and a progressive lesion occurs at each site where the unhampered bacilli have been carried. There are only three ways in which extensive nonencapsulated pulmonary lesions can be produced in the child: (1) by the sudden discharge of large numbers of bacilli from a caseous lymph node into a bronchus; (2) by the primary aspiration of virulent bacilli from the outside, which then proceed to produce a progressive lesion in the nonresistant tissue, and (3) by the lodging of a few virulent bacilli in the lung, followed by a delay in the development of resistance sufficiently long to permit the bacilli to increase greatly in numbers before resistance appears.

The reaction of the adult to tuberculous infection is discussed, and the city-dwelling adult, who has been repeatedly exposed to the bacillus from early childhood onward, is compared to the animal to which a primary protective inoculation of bacilli has been given which has resulted in an encapsulated localized lesion. Because of this localized infection, the body of the adult, just as that of the experimental animal, is allergic, i.e., the product of the bacillus produces necrosis and inflammation of the tissues more readily; it possesses acquired resistance, i.e., the bacilli can not thrive as well in it, and the bacilli appear to be held "fixed" wherever they lodge, so that the spread of viable organisms is delayed and greatly hampered.

The authors discuss the origin of adult tuberculosis and do not think that there is evidence that it develops from the original childhood infection, but that it is a reinfection — whether endogenous or exogenous they are unable to say.

Two factors are considered important in determining whether or not miliary tuberculosis will develop: (1) the factor of acquired resistance and (2) the degree of infection of the blood stream. The child is more likely to develop miliary tuberculosis than the adult because: (1) the tuberculosis may become generalized before a maximal degree of resistance has had time to develop, and (2) the more rapid extension of the lesions in the child favors more rapid and extensive infection of the blood stream.

The authors sum up their attitude as to the difference between progressive tuberculosis in the adult and that in the child thus: "In the child bacilli lodging locally grow freely in the body without resistance; many are swept early, in

viable condition, to lymph nodes, and if in sufficient numbers and of sufficient virulence, can grow unchecked and attain to numbers large enough to be too strong for resistance when it at length appears. With the development of allergy in the presence of large numbers of proliferating bacilli, progressive caseous lesions in lymph nodes and elsewhere, advancing too rapidly to permit much repair become prominent. Progressive lesions in almost all city-dwelling adults develop under the influence of some degree of resistance acquired through the primary and subsequent infections, and they progress more slowly because the growth of bacilli is retarded by this resistance. Because of the slow extension of tissue damage the opportunity for repair is greater. Viable bacilli are swept to nodes less freely because of the mechanism which, in the resistant body, apparently tends to "fix" them at their site of lodgement, and they grow there poorly because they lodge in resistant tissue. . . . The explanation of the difference between adult and childhood tuberculosis lies in the events which happen in the child *before the appearance of acquired resistance.*"

LYTTLE, New York.

GUMMATOUS OSTITIS OF UPPER END OF FIBULA. E. E. SHATTOCK, Proc. Roy. Soc. Med. **22**:1031 (July) 1929.

A case was reported in a girl, aged 4 years.

WILLIAMSON, New Orleans.

ROENTGENOGRAPHY IN INCIPIENT FORMS OF PULMONARY TUBERCULOSIS IN INFANCY WITH ANATOMIC CONTROLS. P. ARMAND-DELILLE and C. LESTOCQUOY, Bull. Soc. de pédiat. de Paris **27**:92 (Jan.) 1929.

The authors present the roentgenologic plates and corresponding sections of the lungs removed at necropsy from infants who died of pulmonary tuberculosis. They believe that generalized lesions are always extensions from some localized primary pulmonary focus. M. H. Barbier, in a lengthy discussion, takes issue with this view.

BENJAMIN, Montreal.

UNILATERAL OPTIC ATROPHY ASSOCIATED WITH CONGENITAL SYPHILIS. L. BABONNEIX and J. DELARUE, Bull. Soc. de pédiat. de Paris, May, 1929, no. 5, p. 235.

A boy, aged 7 years, was accidentally discovered to be blind in the right eye. There were right-sided optic atrophy, asymmetry of the skull and a scar caused by the forceps in the right temporal region. The Wassermann reaction of the blood was negative. Apparently, the spinal fluid was not examined. The mother had interstitial keratitis and a positive Wassermann reaction.

CLAUSEN, Rochester, N. Y.

THE RUPTURE OF COLD ABSCESSES IN POTT'S DISEASE INTO THE PLEURO-PULMONARY PASSAGES AND INTO THE DIGESTIVE TRACT. E. SORREL and MADAME SORREL-DÉJERINE, Bull. Soc. de pédiat. de Paris, May, 1929, no. 5, p. 232.

In spite of the supposedly good prognosis in cases of rupture of a cold abscess into the pleuropulmonary passages, four of five of the authors' patients died of this complication. On the other hand, they have observed recovery in more than half of their cases of rupture into the pharynx.

CLAUSEN, Rochester, N. Y.

THE DIAGNOSIS OF CONGENITAL SYPHILIS BY THE LANGE REACTION. A. CASTELLANOS, Bol. Soc. cubaña de pediat. **1**:358 (June) 1929.

Castellanos found that the Lange reaction was influenced greatly by the p_H of the solution in the final concentration. This was therefore standardized to neutral in each case. He terms this the "individual Lange." Using this method

in 3,000 cases of congenital syphilis, he found the reaction to be positive in 95 per cent. This reaction appears much earlier than the Wassermann, but on account of the great variation in the precipitations that may occur unless the technic is standardized, the readings require considerable experience.

SANFORD, Chicago.

TREATMENT FOR CONGENITAL SYPHILIS. T. VALLEDOR, Bol. Soc. cubaña de pediat. **1**:384 (June) 1929.

Valledor believes that in congenital syphilis the early manifestations are almost septic. Therefore, it is necessary to give an intensive treatment at this time if injurious results to the organism are to be avoided. Intravenous injections were found to be superior in result to subcutaneous or intramuscular. Likewise, neoarsphenamine was found to be the most efficient arsenical.

SANFORD, Chicago.

PRIMARY TONSILLAR TUBERCULOSIS. ALFONS DROESLER, Monatschr. f. Kinderh. **43**:240 (May) 1929.

The writer reviews previous cases of primary tuberculosis of the tonsil and then reports one of his own. The child, aged 1½ years, was brought to the hospital with pneumonia and died before the case was thoroughly studied. Autopsy showed a primary tonsillar tuberculosis as well as tuberculosis of the submaxillary lymph nodes. The pneumonia was not tuberculous.

GERSTLEY, Chicago.

DESTRUCTIVE CHANGES IN THE BONY SHAFTS IN CONGENITAL SYPHILIS. BERTHOLD EPSTEIN and ERNST PODVINEC, Monatschr. f. Kinderh. **43**:397 (June) 1929.

The classic studies of the last twenty years have distinguished two types of bony change in congenital syphilis: osteochondritis and syphilitic periostitis.

In the last three years the authors studied eighty-six cases, and they submit reports and x-ray pictures showing that changes in the body shafts are frequent; i.e., in 17 per cent of their cases. The observation occurs as a central or as a marginal area of rarefaction. The marginal form is twice as frequent as the central.

Such frequency of involvement of the diaphysis could not have escaped the keen older observers. The authors think that this is a rather new development and is in keeping with the changing manifestations of congenital syphilis. Syphilis seems more visceral in type than it used to be, and with greater visceral involvement will come greater bony involvement.

GERSTLEY, Chicago.

THE MALARIAL TREATMENT OF CONGENITAL SYPHILIS IN CHILDREN. SALA WEITZ, Monatschr. f. Kinderh. **43**:255 (May) 1929.

In five cases the author tried the malarial treatment in congenital cerebrospinal syphilis. Injections of from 2 to 3 cc. of malarial blood were given, and the patients were allowed from seven to twelve chills. The author considers his results better than those with the mercury-bismuth-arsphenamine treatment alone. At any rate, the serologic reactions were improved, but there was not much response clinically. He believes the younger the child the more effective the treatment.

GERSTLEY, Chicago.

Internal Diseases

TULAREMIA. HIRAM E. MILLER and LAWRENCE R. TAUSSIG, Arch. Dermat. & Syph. **19**:378 (March) 1929.

A case of tularemia is reported in which the infection was contracted from a "deer fly" bite. The literature is reviewed and tularemia is discussed in detail.

SHIRLEY, Iowa City.

SOME OF THE CAUSES OF DIFFICULT, NOISY AND RAPID RESPIRATION IN INFANCY AND EARLY CHILDHOOD. JOHN LOVETT MORSE, M. Clin. North America **13**:41 (July) 1929.

Adenoids are the most common cause of difficult respiration in infancy and early childhood. The degree of disturbance varies according to whether the tissue is inflamed and swollen or not. A frequent but avoidable error is to attribute respiratory embarrassment resulting from this cause to other conditions, especially to thymic enlargement.

The symptoms of hypertrophy of the thymus are chiefly noisy inspiration and expiration, continuous or intermittent dyspnea, intercostal retraction and cyanosis, increased by extension of the head. When large enough to cause symptoms, the thymus gland is almost always palpable above the suprasternal notch, and there will be dulness on percussion over the manubrium. The larynx is not depressed during inspiration. Diagnosis of pathologic degrees of hypertrophy should always be possible without x-rays.

Congenital laryngeal stridor is caused by narrowing and infolding of the epiglottis with laxness or congenital elongation of the aryepiglottidean folds. This gives rise to a crowing sound on inspiration that is never evident on expiration. This sound is constant, but varies in intensity with the depth of respiration.

With cerebral hemorrhage, atelectasis of the lungs is the most common cause of disturbance of respiration during the first few days of life. The symptoms, rapid respiration and cyanosis, may be delayed for several days or may be absent entirely. They may be continuous or intermittent, and are increased by crying or nursing. The physical signs are unreliable. Roentgen examination gives the only trustworthy information.

While not the most prominent indication of cerebral hemorrhage, irregularity of respiration is often pronounced and may be the first symptom to appear. Breathing is never noisy.

Rapid and irregular respiration in premature and feeble infants is due in part to imperfect development of the pulmonary alveoli and in part to muscular weakness. Incomplete development of the respiratory centers may be present in premature infants.

The presence of new growths in the upper respiratory tract may be missed if digital exploration is not practiced. The symptoms of congenital atresia of the esophagus are at first largely respiratory on account of the collection of fluids in the nasopharynx. Malformations or paralysis of the diaphragm may cause dyspnea and cyanosis. Physical and roentgen examination will reveal the condition.

Weakness of the muscles of respiration due to amyotonia congenita results in feeble crying, rapid diaphragmatic breathing and later to deformities of the chest. The disease is of rare occurrence and is often not recognized during the first few weeks or months of life; hence, the respiratory difficulties are likely for a time to be erroneously attributed to other causes.

Congenital malformations of the heart are a frequent cause of dyspnea, with or without cyanosis. Idiopathic hypertrophy of the heart, a rare condition, may cause dyspnea by compression of the lungs and interference with the mobility of the chest. A common error is to mistake this abnormality for pleural effusion.

Idiocy, especially of the mongolian type, is often accompanied by continuous irregular or peculiar respiration. Adenoids or slight malformations of the nasopharynx or palate, which are frequently found in these conditions, increase the respiratory difficulty.

Rickets is probably the most common cause of continuously rapid respiration in infancy.

Weakness or paralysis of the muscles of respiration or of the diaphragm occasionally cause continuously rapid or abnormal respiration.

In laryngitis the dyspnea is always inspiratory and is accompanied by noisy respiration, hoarseness or aphonia.

Especially in acute streptococcus infections, there may be inspiratory and expiratory dyspnea. Inspiration is not noisy and the voice remains clear.

Retropharyngeal abscess is often overlooked because digital exploration of the throat is not practiced.

Asthma is a frequent cause of dyspnea, even in early life. It is characterized by expiratory difficulty and the usual physical signs of the condition.

In "breath-holding" and laryngismus stridulus, the characteristic sequence of events is: several short inspirations accompanied by a crowing sound; sudden stopping of respiration on inspiration; cyanosis, occasionally terminating in a convulsion; relaxation of the spasm, and resumption of respiration.

New growths in the larynx give rise to noisy, difficult respiration, chiefly inspiratory, with changes in the voice.

There is nothing pathognomonic about the respiratory disturbances occasioned by foreign bodies in the respiratory tract, although paroxysms of coughing are suggestive. The physical signs in the chest are almost always unilateral.

Mediastinal tumors are likely to give rise to continuous chronic disturbances of respiration, with usually more trouble with inspiration than with expiration.

Pressure on the trachea may cause dyspnea and coughing. Respiration may be noisy, "wheezy" or quiet, and there is usually both inspiratory and expiratory difficulty.

In infancy the earliest and most marked symptom of meningitis, especially the pneumococcus type, may be rapid respiration.

Among other conditions which may give rise to disturbances of respiration in early life may be mentioned suprarenal hemorrhage of the new-born, pericarditis, chronic bronchopneumonia, chronic interstitial pneumonia, mediastinitis and many conditions within the abdomen. At first thought, it would seem from the number of possible causes of difficult, rapid and noisy respiration, that the diagnosis would be difficult. However, the symptoms of many are pathognomonic. While the others have many symptoms in common, there are enough differences in the histories and physical signs which they present to make the diagnosis between them reasonably simple, provided these diseases are kept in mind and reasonable care is exercised.

SMITH, Ogden, Utah.

PYLOROSPASM AND CARDIOSPASM IN AN INFANT. LEWIS WEBB HILL, M. Clin. North America **13**:173 (July) 1929.

Hill reports the occurrence of cardiospasm in a child, aged 2 years, which became manifest at about the age of 6 months. Previously he had suffered from pylorospasm, with vomiting and visible peristalsis, which yielded to thick gruel feedings. A little later, while still on a liquid diet, his appetite failed and he began to spit up a good deal, but vomited only occasionally. After this condition had persisted for some months, to please the mother a roentgenogram was taken, which showed spasm or stricture of the esophagus near the cardiac end of the stomach, with slight dilatation. The stomach emptied normally. Much relief was obtained following esophageal dilatation, but the condition had a tendency to recur.

After one and one-half years, there were symptoms and roentgenologic evidences of cardiospasm, although organic stricture could almost certainly be ruled out by the slight degree of dilatation of the esophagus.

These cases are rare in infancy and hence are likely to be overlooked. Organic stenosis of the esophagus does not manifest itself until solid food is taken, unless there is complete or almost complete atresia. Then it will be noticed that milk goes down well but solid food does not. Roentgenograms should always be taken when an infant exhibits these symptoms.

SMITH, Ogden, Utah.

CHRONIC PYURIA IN INFANCY AND CHILDHOOD. MENDETH and CAMPBELL, M. J. & Rec. **130**:90 (July 17) 1929.

Attention is directed to the frequency with which the diagnosis of chronic pyelitis is made in young patients showing a persistent pyuria. Complete urologic examination of patients suffering from chronic pyuria of more than four weeks'

duration in spite of intensive medical treatment, usually reveals gross urinary lesions which require other than medical treatment. In many of these conditions the administration of a urinary antiseptic is of invaluable aid, while the indicated technical surgical treatment is being carried on; such antiseptics used alone, however, are rarely of permanent curative value. Most chronic pyurias originating in the urinary tract result from obstruction with urinary stasis. Removal of the obstruction in these cases constitutes the cure. With the persistence of the obstruction, severe renal damage and infection may be anticipated. Neuromuscular disturbances of the urinary tract are a frequent cause of chronic pyuria and usually lead to the erroneous diagnosis of chronic pyelitis. Pyuria of genital origin is frequently observed; when the site of the pus in these cases is not generally recognized, the incorrect diagnosis of chronic pyelitis is most often made. Children withstand urologic examination admirably, manifesting less fever and less severe postinstrumental reaction than do adults. Therefore, when the indications outlined exist, such an examination should be urged and carried out. When competently performed, these examinations will strikingly demonstrate not only the incorrectness of the diagnosis of chronic pyelitis on the basis of persistent pyuria, but also the curious and numerous lesions of the urinary tract which may bring about this chronic pyuria. It will be found that (1) uncomplicated chronic inflammation of the renal pelvis does not exist, (2) pus originating in the upper tract is, in the main, from the renal parenchyma and (3) in many instances, involvement of the upper tract is meager or absent, while disease of the lower urinary tract is marked.

WILLIAMSON, New Orleans.

OBSCURE FEVER IN CHILDREN. J. EPSTEIN, M. J. & Rec. **130**:92 (July 17) 1929.

A rise in temperature is usually the first evidence of some abnormality in the processes of the human organism, and infants and children may be the only evidence of a pathologic process. The following are some of the causes of protracted fever in children with delayed signs and symptoms: (1) febris neonatorum, (2) sepis, (3) congenital syphilis, (4) rickets and scurvy, (5) influenza, (6) malaria, (7) pyelitis, (8) endocarditis, (9) tuberculosis, (10) typhoid, (11) pulmonary sepsis, (12) blood diseases, (13) adenitis, (14) chronic otitis, (15) general pneumococcus or meningococcus infection, (16) external heat and (17) pyretic drugs.

Fever is a defensive reaction of the body against disease, and subsides when the reparative processes of the body have mended the injury. Fever itself requires no treatment with drugs. When a diagnosis is made, the treatment is that for the underlying disease.

WILLIAMSON, New Orleans.

EXTENSIVE VISCERAL ENDARTERITIS OBLITERANS IN A YOUNG CHILD. JOHN MCMICHAEL, Arch. Dis. Childhood **4**:165 (Aug.) 1929.

A case report is submitted of an English child, aged 18 months, who died of extensive thrombo-angiitis obliterans. The most noticeable lesions were those of endarteritis obliterans of the small arteries. There was great connective tissue proliferation internal to the internal elastic lamina. There was no evidence of syphilis.

PETERMAN, Milwaukee.

STUDIES OF PNEUMONIA IN CHILDHOOD. CHARLES MCNEIL, AGNES R. MACGREGOR and W. ALISTER ALEXANDER, Arch. Dis. Childhood **4**:170 (Aug.) 1929.

This is the fourth report of an exhaustive study of pneumonia in childhood. The authors are making a worthwhile contribution. Their studies are extensively illustrated with good sections. It is suggested that the majority of cases of bronchiectasis date back to early childhood and originate in bronchopneumonia or bronchitis. The first stage of the process is the interstitial inflammation of the

bronchial walls, which goes on to necrosis and suppuration. A cavity is formed by the lines of tissue and excavation of the adjacent alveolar substance. The cavity may be cylindric or saccular. Subsequently it is lined with granulation tissue which becomes fibrous and may finally be covered by bronchial epithelium. Consequently a bronchiectatic cavity is not a dilatation but an excavation. Fibrosis of the lungs may be considered the result of long continued interstitial inflammation of the lung framework.

PETERMAN, Milwaukee.

DISPLACEMENT OF THE HEART IN PNEUMONIA IN CHILDHOOD. KENNETH H. TALLERMAN and MONTAGUE H. JUPE, Arch. Dis. Childhood **4**:230 (Aug.) 1929.

Five cases are reported. It is suggested that the displacement of the heart is caused by the traction of the partially collapsed lung and the pressure of the sound lung distended by compensatory emphysema.

PETERMAN, Milwaukee.

PNEUMONIA, EMPYEMIA, NEPHRITIS, AND PNEUMOCOCCAL PERITONITIS WITH COMPLETE RECOVERY. A. G. MAITLAND JONES, Proc. Roy. Soc. Med. **29**:659 (March) 1929.

A patient, aged 5 years, was admitted to the hospital with a history of swelling of the glands in the neck, rapid respiration and fever at night. Examination showed impairment of the percussion note and decreased breath sounds at the left lower lobe; the urine contained albumin, blood cells, granular and hyaline casts. The temperature was 103 F. Pain in the abdomen commenced on the fifth day; a diagnosis of pneumococcal peritonitis was made, and a suprapubic drainage was carried out. The condition improved and the child was sent to a convalescent home where the temperature was found to be elevated at night. An exploratory examination was made, and pus was found over right lower lobe. Rib resection was carried out, and the patient made an uninterrupted recovery. The urine cleared up. On reexamination a month after discharge from the hospital, no abnormal physical signs were found.

WILLIAMSON, New Orleans.

TWO CASES OF CELIAC INFANTILISM. H. GARDINER-HILL, Proc. Roy. Soc. Med. **22**:1033 (June) 1929.

Two cases of celiac infantilism were reported in girls, aged 22 and 15 years, respectively.

WILLIAMSON, New Orleans.

PNEUMOCOCCUS PERITONITIS IN INFANTS. P. MATHIEU and J. DAVIOUD, Presse Méd. **93**:911 (July 13) 1929.

Female infants with a high temperature, a rapid pulse rate and a peritoneal syndrome accompanied by an abundant green diarrhea may have pneumococcus peritonitis. The case may be diagnosed as acute appendicitis and the abdomen opened. If this is done, the green pus in the lower part of the abdomen will show the condition. Drainage and treatment for septicemia are given. In the presence of the foregoing symptoms, the condition should always be suspected and should be confirmed by bacteriologic examination. In these cases it is better to wait until the pus localizes before surgical intervention. It may not be necessary to intervene in many cases.

SANFORD, Chicago.

THE PATHOGENESIS OF "CIRCULAR CARIES" OF THE DECIDUOUS TEETH. CATERINA DESSYLLA, Riv. di clin. pediat. **27**:401 (May 30) 1929.

"Circular caries" of the deciduous teeth occurs around the gum margins of the four upper incisors and is frequently found in young children. Acquired or congenital failure of the intermaxillary bone to develop or to be nourished properly

is a predisposing factor. Deformity of the mouth from adenoids is thus a common predisposing factor. Usually one of the general diseases of childhood, such as rickets, syphilis, tuberculosis or a respiratory infection is a contributing factor in causing this type of dental caries in a child predisposed by oral abnormality.

HIGGINS, Boston.

ALTERATIONS OF THE MYOCARDIUM IN THE INFECTIOUS DISEASES OF CHILDREN. E. MOLINARI, Riv. di clin. pediat. **27**:442 (May 30) 1929.

In a thesis written as part of the author's university work the literature is reviewed and studies of the heart in fifteen autopsies are given.

Two types of myocardial changes are found in the infectious diseases — degenerative and inflammatory. The degenerative change is found in the toxic diseases, such as diphtheria and gastro-enteritis.

HIGGINS, Boston.

TREATMENT OF PYELONEPHRITIS IN INFANCY. C. BARROSO, Arch. de pediat., Rio de Janeiro **2**:361 (Aug.) 1929.

An autogenous vaccine is made at once from organisms cultured from the child's urine. Two or more injections of this are given about three days apart. If the child's condition is quite extreme, from 50 to 100 cc. of blood is given as a transfusion, followed by from 20 to 30 cc. intramuscularly.

SANFORD, Chicago.

BRONCHIAL ASTHMA IN EARLY CHILDHOOD. K. STOLTE, Jahrb. f. Kinderh. **122**:1 (Nov.) 1928.

Stolte reports a series of six cases of bronchial asthma in young children ranging in age from 7 weeks to 3 years. The onset is quite sudden and may occur suddenly or may be preceded by catarrhal manifestations. There is marked dyspnea, severe straining of all the accessory muscles of inspiration with marked suprasternal and epigastric retraction, inability to perform complete expiration, and deep cyanosis of the lips and extremities. There is a severe pallor of the body, and a puffy, bloated face. The attacks may simulate cardiac decompensation, laryngeal diphtheria or foreign stenosis; the thorax is markedly distended. On auscultation, there is a weakening of breath sounds with a prolonged expirium. The distention of the lung may be so marked as to extend over the heart area and obliterate the area of cardiac dulness on percussion.

Heubner notes that "asthmatic bronchitis" is frequent and may occur even in children from 6 to 10 weeks of age. It may occur without previous catarrhal manifestations. Spasm and swelling of the mucosa is sufficient to cause obstruction of the narrow bronchioles. Because of the particularly narrow bronchioles in infants, these attacks may threaten the life of the patient.

There is no unified therapy. Narcotics produce good results. Ehle recommends urethane. The author administered oxygen inhalation and calcium (5 cc. of calcium afenil intravenously or 10 cc. of calcium Sandoz intragluteally).

ABT, Chicago.

VOLHARD'S STASIS FOLLOWING CONCRETIO CORDIS IN CHILDHOOD. W. LENGSFELD, Jahrb. f. Kinderh. **122**:95 (Nov.) 1928.

As Lengsfeld notes, it is well known that the pericarditides and their sequelae are more frequent in childhood than they are diagnosed. The author describes the customary manifestations of adhesive pericarditis and the various symptoms.

Systolic retraction of the thoracic wall is considered the chief symptom of adhesive pericarditis. However, in this instance the author reports a case of concretio cordis and gives credit to Volhard for having first clarified the syndrome. In this instance there is not the usual polyserositis or adhesive pericarditis, but the

pericardium becomes scarred and fixed, interfering with the proper diastole but not to any considerable extent with the systole of the heart. This leads to a stasis, hepatic enlargement, ascites and dropsy. On physical examination, the heart itself exhibits no murmurs or abnormalities. Medical therapy, such as urea and mercurial salts, is merely palliative in relieving stasis. The treatment of choice is operative and consists of pericardiolysis, as recommended by Schmieden and Oelorme. The etiology is usually rheumatic or tuberculous pericarditis.

ABT, Chicago.

ROENTGEN TREATMENT FOR PYLOROSPASM IN INFANTS. CARL WIENER, Jahrb. f. Kinderh. **122**:113 (Nov.) 1928.

The author believes in a restriction of operative indications in the treatment for pyloric stenosis. He indicates the good results of purely medicinal and dietary regulation and quotes such authors as Czerny-Keller, Ibrahim and Rietschel who believe in restricting the indications for operative management.

He reports six cases of pyloric stenosis in which he used x-rays and in which the patients apparently made satisfactory recoveries. The treatment consisted of 10 per cent skin unit dosage through a 3 or 4 mm. aluminium filter at a distance of 40 cm. and directed at the pyloric region. After the treatment there was a sudden stop in the vomiting, and the usual diet could then be employed.

ABT, Chicago.

THE MANAGEMENT OF JUVENILE DIABETES. R. PRIESEL and R. WAGNER, Wien. klin. Wchnschr. **42**:1170 (Sept. 5) 1929.

The thirty children with diabetes who were observed at the Pirquet clinic before insulin became available in 1922 have all eventually died. Only one of the ninety-four admitted since 1922 has died; his parents refused to carry out the prescribed treatment. The authors describe in detail their method of keeping in contact with their patients after a careful study in the hospital has been completed. Parents are not permitted to make examinations of the urine. Patients return to the outpatient clinic at regular intervals of one or two weeks. Special efforts are made to have these children properly educated. As long as they receive suitable diet and proper doses of insulin, they are regarded as normal, and may indulge in sports. The authors have succeeded in getting the government to furnish insulin free of charge as long as this is necessary. At the age of 14, the patients who must earn their living are enrolled in a "Krankenklasse," and are taught a trade.

CLAUSEN, Rochester, N. Y.

CYST FORMATION IN THE LUNGS OF CHILDREN. H. VOLLMER, Ztschr. f. Kinderh. **46**:810 (Dec. 15) 1928.

A child, aged 5½ years, became ill with fever; though the fever came down in twelve days, she still coughed and looked sick. Four months later, a roentgen examination showed an infiltration in the right midfield. The tuberculin test gave a negative reaction; the red corpuscle precipitation reaction was normal, and there was no shifting of the blood picture. Two months later, another roentgenogram showed a cyst with its capsule, and one and one-half months later the picture was that of an atypical cavity with fluid, possibly, except that the child seemed too well. The tuberculin test, repeated six times, continued to be negative. There must have been a nonspecific cavity which arose from the resolution of a post-pneumonic residual infiltration.

An infant, aged 10 months, was admitted with a history of having had cough, vomiting and night sweats for four months, and an attack of coughing, fever, dyspnea and cyanosis three months before, which was thought to be pneumonia because the fever went down, though subsequently it rose to 100.4 F. and remained there for weeks. The patient appeared very sick, pale, dyspneic and mildly cyanotic on admission, and showed retraction of the ali nasi and of the epigastrium. The whole right lung was tympanitic to percussion and showed diminished breathing.

The reaction to the tuberculin test was negative. Roentgen examination showed a large air-containing sac in the form of an egg in the right side of the thorax, moving with respiration and reaching from the apex to the cardiophrenic angle. The lung tissue outside the cyst was thickened but not collapsed, and the heart and mediastinum were pushed to the left. On the introduction of a needle, air escaped with a whistling noise; the child breathed with difficulty, was restless and looked pale for a short time but quickly recovered. From then on, the breathing became quiet and regular. The following day the child laughed for the first time in weeks. Two days later, there remained a little dulness and diminished breathing; roentgen examination showed a diffuse shadow of the right lung, but the patient's general condition was good. It seemed that there must have been an interstitial transudate following the sudden release of compression. After four more days, the shadow disappeared. The probable diagnosis was an air rupture in a peripheral portion of the lung tissue: that is, a pneumatocele of unknown origin.

FIBROPLASTIC APPENDICOTYPHLITIS. BOHUSLAV VOZENILEK, Časop. lék. česk. **65**:2000 (Dec. 24) 1926.

A boy, aged 14, who had been treated for tuberculous peritonitis, was found to have fibroplastic inflammation of the appendix, cecum and ascending colon. Macroscopically, there was an induration and thickening of the bowel wall, progressing from the appendix. Microscopically, this was found to be a lymphocytic and leukocytic infiltration, especially of the subserous layers. No tuberculous involvement of the glands was noted. The etiology is obscure. To date, fifty-one cases have been reported. The differential diagnosis includes tuberculosis. The treatment is radical excision of the involved bowel, usually the terminal ileum, appendix, cecum, the ascending and even the transverse colon.

STULIK, Chicago.

TREATMENT OF MYXEDEMA IN CHILDREN WITH PREPARATIONS OF THE THYROID GLAND. A. KIESEL, Med.-biol. j. **4**:20, 1928.

The author confines himself to myxedema because it is a disorder of the thyroid gland which is most frequently encountered in children and because it is most familiar to the author. Altogether, 100 children with myxedema were studied and observed. The work began in 1898, and some patients were observed for from several to thirty years. In children from the ages of 5 and 15, the daily dose of thyroid extract should not exceed 0.12 Gm. Larger doses cause symptoms of intoxication with a rapid pulse, flushing of the face, restlessness, epigastric pains, nausea, diarrhea, and occasionally a rise in temperature. In small children, 0.06 Gm. of thyroid is sufficient.

As soon as equilibrium is established with improvement in mental and physical symptoms, half of the previous dose should be given for the rest of the time. The author cites several cases, of girls previously sluggish and underdeveloped, in which administration of thyroid resulted in intellectual improvement as well as the appearance of secondary sexual characteristics. It is much better to keep on giving small doses of thyroid all the time. Periods of rest from the drug were found by the author to be unsatisfactory in the sense of appearance of previous symptoms.

KASANIN, Boston.

Nervous Diseases

STUDIES IN STUTTERING: V. A STUDY OF SIMULTANEOUS ANTITROPIC MOVEMENTS OF THE HANDS OF STUTTERERS. L. E. TRAVIS and R. Y. HERREN, Arch. Neurol. & Psychiat. **22**:487 (Sept.) 1929.

Right-handed normal speakers with a history of right-handedness lead more frequently with the left hand; right-handed normal speakers with a history of left-handedness lead about as often with one hand as with the other; left-handed

normal speakers with a history of left-handedness lead more frequently with the right hand, and right-handed stutterers, regardless of a positive or a negative history of left-handedness, lead more frequently with the right hand. No significant differences seemed to exist between the groups, relative to simultaneous leads.

SANFORD, Chicago.

THE EARLY DIAGNOSIS OF MENTAL DEFECT. ARNOLD GESELL, Arch. Neurol. & Psychiat. **22**:522 (Sept.) 1929.

Gesell gives three cases to illustrate the importance of small amounts of deviation in the diagnosis of development in early infancy. There is a spurious degree of retardation in the premature infant; the chronologic age is therefore so consequential that careful clinical discount must be made for the interval of prematurity. Even severe physical handicaps do not drastically alter the behavior capacities of the child. In its most characteristic form, mental deficiency does not come through abrupt abbreviation or sharp curtailment of normal growth. It seems rather to express a fundamental, pervasive reduction of growth potency. When the fate of the person rests on such a reduction, a diagnosis of mental defect may be safely made in early infancy.

SANFORD, Chicago.

A HAEMANGIOMA OF THE MENINGES INVOLVING THE VISUAL CORTEX. R. F. MOORE, Brit. J. Ophth. **13**:5 (May) 1929.

A boy, aged 12 years, complained only of defective vision of his left eye. Three years before, vision in this eye was 6/12. It had grown worse in the past year. A nevus of the left half of the face involved the eyelids. At the time of presentation, vision in the right eye equaled 6/9, partly, and that of the left 6/6, partly. There were an incomplete right homonymous hemianopia, a slight congenital dislocation of the lenses and a small coloboma of the left optic disk. A diagnosis of nevus of the visual cortex was made. This was confirmed by the roentgenogram which showed a tumor composed of a new growth of large vessels. A lateral roentgenogram showed the growth to be acutely wedge-shaped, the apex being far forward in the temporal region and apparently being situated superficially under the bone in this region. The base of the wedge was at the occipital pale immediately above the tentorium and was therefore closely related to the visual cortex. The dense shadow was probably due to calcification of the vessel walls. Mention is made of a number of reported cases of this kind. Buphthalmos is often associated, as is also heterochromia iridis. Lesser anomalies, such as occurred in the present case, are encountered. The ocular abnormality, when unilateral, in all instances was on the same side as the tumor of the brain. The author advises that in all cases of buphthalmos a roentgenogram of the skull be taken. He repeats a suggestion made by him several years ago that in cases of melanoma of the choroid one should watch for intracranial tumor.

W. ZENTMAYER.

THE CHOLESTEROL OF THE BLOOD PLASMA IN EPILEPSY. MINNIE GOSDEN, J. TYLOR FOX and W. RUSSELL BRAIN, Lancet **2**:12 (July 6) 1929.

This study is based on observations made on twenty-one epileptic patients and five controls. A modification of Bloor's method was used. Most of the observations were made during the night with the patients at rest in bed and several hours after the last meal. All the patients studied were clinically healthy except for the fits. The average blood cholesterol in this group of epileptic patients was found to be below normal. The authors conclude that status epilepticus and serial epilepsy are associated with especially marked depression of the blood cholesterol. The extremely low figure of 0.036 per cent was obtained in one patient at the end of a severe attack of status epilepticus.

The authors state: "In our present state of ignorance concerning both the nature of a convulsion and the metabolism of cholesterol we can offer no opinion as to the nature of the association between changes in the blood cholesterol and the occurrence of fits."

LANGMANN, New York.

STUDIES OF A HUNTINGTON FAMILY. (A CONTRIBUTION TO THE SYMPTOMATOLOGY OF DIFFERENT STAGES OF HUNTINGTON'S CHOREA.) O. REISCH, Arch. f. Psychiat. **86**:327 (Feb.) 1929.

The author reports a study of five generations of the family of a patient whose case had been reported previously *(Arch. f. Psychiat.* **74**:795, 1926). The purpose was to determine the early symptoms of the disease, before it is definitely established, and also the possibility of the occurrence of abortive syndromes. The cases of definite chorea in this family are reported in detail. Brief summaries are also given of cases which were probably milder, and also of cases in the fifth generation which showed signs especially in the motor system that would point to the possibility of future development of the disease. This is substantiated by the fact that similar symptoms were elicited in the histories of patients in whom the disease has subsequently developed.

Reisch summarizes his observations as follows: Two sisters of the patient previously reported showed symptoms that were essentially those of Huntington's chorea, especially the muscular tension phenomena. These phenomena, although not advanced to the stage of a general rigidity, manifested themselves in intermittent muscle spasms during passive movement. Similar "tension phenomena" were also found in four young members of this family and were accompanied by mild character changes, motor clumsiness and occasional spontaneous movements. Since such symptoms were also observed in the early stages of definite chorea, they may serve as useful early indications of this disease.

MALAMUD, Foxborough, Mass.

THE WHITE BLOOD CELL PICTURE AND THE CAPILLARIES OF THE NAIL GROOVE IN YOUNG MENTAL DEFECTIVES: SECOND COMMUNICATION ON THE SHAPE OF THE CAPILLARIES IN THE FEEBLEMINDED. H. K. KAHLE, Arch. f. Psychiat. **86**:766 (March) 1929.

In this paper the author reports further results in the investigation of the relationship between physical deviations and feeblemindedness. Three hundred and forty-three mentally defective children were examined. The white blood cell counts and differential examinations, as well as the shape of the capillaries of the nail groove, were particularly studied. It was found that the average case showed an increased number of white blood cells with a relative lymphocytosis and eosinophilia. This was true in most cases, although it was not found in some special groups such as the mongoloid idiots, those with hypothyroidism, the hypoplastic subjects and the dwarfs. The shapes of the capillaries were found to be fairly characteristic and as follows: In the main group of congenital feeblemindedness (in the District of Hanover) the so-called archiocorrected shapes or pseudoneo-capillaries of Hoebfner were found without any tendency toward productive formations. The fact that the shapes of the capillaries are related to certain types of idiocy was made use of in outlining a treatment, especially for that of endocrine deficiencies. Certain small groups of the feebleminded could be differentiated from the main group by the shapes of the capillaries; this is especially so in the case of idiocy due to organic diseases, the athetoid types and those with suprarenal disturbances. The author concludes with a statement that further investigation into the nature of the physical phenomena accompanying mental deviations may be useful in outlining the management of such cases and in determining just what form of training should be instituted in special types. This would also help in attempts at treatment, glandular as well as chemical. The author used tincture of iodine and different glandular preparations. In some cases in which there was a definite indication of the type of endocrine gland involved, that single gland could be used. In other cases, one had to utilize a mixed therapy.

MALAMUD, Iowa City.

THE CARBOHYDRATE METABOLISM IN PROGRESSIVE MUSCULAR DYSTROPHY. M. S. SCHEIMANN, Arch. f. Psychiat. **87**:665 (June) 1929.

The blood sugar was studied in eight cases of progressive muscular dystrophy to determine whether any differences could be found between the carbohydrate metabolism of these patients and that of normal persons. The blood sugar during fasting, with the patient at rest, showed no essential deviations outside of the fact that it was universally low (from 60 to 70 mg.). The blood sugar curve following administration of sugar showed a definite hyperglycemic curve along with some glycosuria. A similar condition was found on a study of the blood sugar following muscular exertion. Both of these observations were similar to those made on diabetic patients. The author considers these results significant in that they show that while these patients exhibit no abnormal metabolism when at rest, their ability to metabolize higher quantities of sugar, or the normal quantities during muscular exertion, are seriously interfered with. He believes that this may give some leads in the study of the pathogenesis of the disease and its etiology. Further study should be undertaken, however, on both the other aspects of carbohydrate metabolism and the metabolism of other substances (proteins, fats, salts, etc.).

MALAMUD, Iowa City.

THE PATHOGENESIS OF INVOLUNTARY MOVEMENTS WITH SPECIAL REFERENCE TO CHOREA. S. A. KINNIER WILSON, Deutsche Ztschr. f. Nervenh. **106**:109 (Dec.) 1928. (From the Proceedings of the Eighteenth Annual Congress of the German Neurological Society held in Hamburg, Sept. 13-15, 1928.)

As Hughlings Jackson's pupil, I cannot be satisfied with the manner with which many neurologists associate clinical symptoms with pathologic lesions. The fundamental mistake of the clinical-pathologic method is that there are two types of clinical symptoms, such as destruction of function and dysinhibition of function; but there is only one kind of pathologic lesion which can be proved with certainty, namely, destruction. Many neurologists believe that choreic movements can be explained by a deficiency in striatum elements. Against this I may say that histologic lesions affecting paralysis can be explained only on the basis of a destruction or loss of nerve function. But the problem of all involuntary movements —as tic, epilepsy, tremor, chorea, athetosis, myoclonus, etc. — is one of physiology. It is clear that these problems can never be settled if one assigns as the cause of these movements a destroyed cell fiber system. All involuntary movements depend on a living mechanism, and the question must therefore not be: Which mechanism is paralyzed? But rather, Which mechanisms are in activity? For the problem of chorea, a careful analysis of the choreic movements is of great importance. One finds that the spontaneous movements in chorea strongly resemble those which are designated as voluntary. They appear as complicated, coordinated and serving a definite end as those of the free selective type. On the other hand, the voluntary movements of the choreic patient are often as short, abrupt and explosive as the involuntary movements. Further, in chorea one finds definite more or less "minor signs" which point to a corticospinal disturbance. From those clinical facts the following conclusions may be drawn: the choreic disturbance manifests itself through the corticospinal (pyramidal) system; this must be in a relatively entire condition, making it possible for the hyperkinetic symptoms to appear. Yet it may show a defect in its function. Therefore, as I observed, the voluntary movements of the choreic patients do have something of the character of involuntary movements; the latter take the same route and usurp at the cost of the former. Wilson showed some pictures in a case of senile chorea which was under his observation for eleven years. The patient, a woman, died at the age of 88. The marked choreic movements affected only the right side. The postmortem observations showed that the convolutions were generally well maintained; the left postcentral convolution alone was shrunken to nearly one-half the size. The entire left corpus striatum was well preserved in all parts, even considering the age of the patient. There was no macroscopic difference between the right and left

striatum. The left putamen showed microscopically as good as normal structure; the large as well as the small cells were intact. According to Wilson this case explodes the striatum theory of chorea. He would consider the atrophy of the left postcentral convolution of the greatest importance for the pathogenesis of chorea, and offered the following hypothesis: for the occurrence of choreic movements it is necessary to have a relatively integral efferent tract. Many compelling reasons suggest the assumption that this tract is the corticospinal. He has never seen a case of chorea or of athetosis in which spontaneous movements took place in association with a complete corticospinal paralysis. Such movements were never observed in man with decerebrate rigidity, and Magnus stated that it was absent in lower animals which he has decerebrated. The explanation which the author would give in the cases of chorea or athetosis is that in decerebration the motor cortex is out of function. A number of clinical facts would indicate that in cases of chorea-athetosis the afferent tracts from the cerebellum via the regio subthalamica to the thalamus and the brain cortex are involved; hence, the great importance of the postcentral atrophy in his case of chorea. Certain changes in the sensorimotor cortex have taken place which point to a disturbance in the regulation of motor stimulation of the transcortical as well as of the cerebellocerebral side. The author points out as the most characteristic trait in chronic choreic movements the inability on the part of the patient to influence voluntarily the movements or to bring them to a standstill: expressed physiologically, it can only indicate a transcortical damage of the function, and in his case he connects the loss of the transcortical control with the atrophy of the sensory part of the cortex. In this place, most likely, the activity of the cerebellocerebral component occurs. The author summarizes his conceptions as follows: chorea and choreic athethosis represent a complex type of involuntary movements for the activity of which a motor mechanism having its seat in the cortex is required. It is not possible to assume that a single circumscribed anatomic area is responsible for developing these symptoms. Chorea is an expression of the disturbance of a system. A defect in the afferent cerebellocerebral regulation contributes to its development; the transcortical (voluntary) is very incomplete in its expression. No theory which assigns a single destructive lesion in the corpus striatum as the cause of chorea will agree with the facts. No hypothesis as to the cause of involuntary movements can be discussed which does not follow the interpretation of semiologic and pathologic principles, thanks for which are due largely to the genius of Hughlings Jackson.

Discussion.—DR. O. MARBURG: I agree with Wilson that chorea and athetosis are complicated processes, but they do not take place only through disturbance in the transcortical regulations. All lesions in the cerebellocerebral tract are capable of producing a similar disturbance without participation of the cortex. I recall a case reported by Halban-Infeld, in which a small circumscribed lesion was responsible for the athetosis, and a case reported by Herz in which a small lesion was found in the thalamus. A lesion in the end-system is also capable of producing the same; fundamentally it is concerned with a centripetal regulation disturbance.

DR. MINGAZZINI: I agree entirely with Wilson when he assumes that choreic movements depend only on disturbance of the activity of the cerebellocerebral (cerebropetal) fibers. For many years I have supported the assumption of the existence of such fiber complexes, especially on the basis of personal investigations by the absence of lacunae in sections of the brain in man, in one of which was found an aplasia of the cerebellum, as well as in those of the dog and monkey in which the cerebellum or the cerebrum was removed and in the human brain when the cerebellum was found nearly absent on one side. Now in these cases the homolateral and the contralateral transverse fibers of the pons, the fibers of the medial and lateral fifth of the pes peduncili, the anterior segment of the internal capsule and some contralateral frontal convolutions were in places either partly degenerated or aplastic. The course of the disease process spoke distinctly for the presence of cerebellofugal (not cerebellopetal) fibers which end in the frontal lobes and perhaps connect with the pyramidal cells in the rolandic area.

These cases agree completely with the pathogenesis of the movements in chorea and athetosis as given by Wilson.

DR. A. WIMMER: I agree entirely with Kinnier Wilson in his demand for the entirety of the corticopyramidal system for the appearance of choreic disturbances. The comparison of choreic and normal movements is not entirely convincing; not only is it a question that the choreic patient cannot voluntarily suppress his movements, but it concerns rather a disturbance of a succession of movements, a disturbance of transmission. I agree with Kinnier Wilson that destruction cannot give positive physiologic disturbances. Things are altogether different when it concerns a possible disturbance of transmission. One must hold to the idea that such disturbances can be effected chiefly from the corpus striatum. At any rate one must at present consider the corpus striatum as the most important "editorial place" in the brain in the bringing on of involuntary movements.

BERNIS, Rochester, N. Y.

THE OCCURRENCE OF PRIMARY PURULENT MENINGITIS WITH THE ACUTE INFECTIONS OF CHILDHOOD. HERMANN ZISCHINSKY, Jahrb. f. Kinderh. **121**:221 (Oct.) 1928.

Hermann Zischinsky studied carefully primary purulent meningitis that occurs as a consequence of acute infection in childhood. The material comprises the case records with autopsy reports from 1902 to 1928. After a short description of the previous literature, the author reports in detail on seventy-two cases in his own material. Of 800 patients with erysipelas, 4 died of purulent meningitis. Of 12,500 patients admitted with diphtheria, 2 died of streptococcus meningitis. There were four instances of streptococcus meningitis occurring after scarlatina. There were six deaths from purulent meningitis among 10,700 patients with measles. However, pertussis furnished the largest number of cases of meningitis. Among 10,900 victims of pertussis, there were 2,550 deaths; 51 were proved at autopsy to be due to purulent meningitis. In several instances there was no evidence of meningeal irritation, and no indications for lumbar puncture.

ABT, Chicago.

THE SYMPTOMS OF MENINGITIS. JOSEPH AMBRUS, Jahrb. f. Kinderh. **121**:249 (Oct.) 1928.

After remarking on the great similarity in the symptoms of meningismus and meningitis, the author describes the causes of these symptoms and a rational method of therapy, particularly in basillar meningitis. The author concludes that Kernig's sign and other symptoms of meningitis are not due to an irritation of the gyrus centralis, but to edema of the brain tissue itself. Another factor in the causation of these symptoms is acidosis, which also is associated with biochemical changes in the tissues and with edema of the brain. The author concludes with an experimental study of pieces of brain tissue immersed in various solutions of varying p_H. He found that edema of the white substance is more extensive than that of the gray substance. Of the various solutions used, sodium sulphate, acetate and citrate, the last had the most marked therapeutic effect in reducing the edema.

ABT, Chicago.

CLINICAL AND EXPERIMENTAL STUDIES OF PHENYLETHYLHYDANTOIN IN THE THERAPY OF CHOREA IN CHILDHOOD. B. LEICHTENTRITT, W. LENGSFELD and SILBERBERG, Jahrb. f. Kinderh. **122**:12 (Nov.) 1928.

In the recent literature various writers, such as Roeder, Rietschel, Schmal and de Rudder, have not had favorable therapeutic effects from medication with phenylethylhydantoin. Leichtentritt, W. Lengsfeld and Silberberg, at the Breslau Pediatric Clinic, report on the use of phenylethylhydantoin in twenty children, aged from 4 to 13 years. The dosage was from 0.2 to 0.45 Gm. three or four times daily,

according to the age of the child. The authors report 80 per cent of patients cured and 20 per cent improved.

The authors consider phenylethylhydantoin a favorable remedy in chorea minor. No unfavorable effects were observed on the cardiovascular system. In a number of cases an exanthem appeared from the seventh to the twelfth day; the rash was sometimes morbilliform or scarlatiniform and frequently urticarial, and was associated with a considerable rise in temperature. There are associated blood changes: leukopenia, relative lymphocytosis, eosinophilia, monocytosis and thrombopenia. This complete "phenylethylhydantoin reaction" (Pfaundler) does not occur in all cases. At times, one sees only partial reactions. The most constant of all symptoms is eosinophilia, which may precede all the other manifestations.

Large doses of phenylethylhydantoin administered to rabbits induce aleukia similar to that induced by benzene, arsphenamine, x-rays, radium and thorium. This should indicate care in therapy. The period of treatment in the human being should not exceed ten or twelve days. Before and during the treatment, the leukocytes of the blood should be carefully checked.

ABT, Chicago.

ADVANCES IN THE METHOD OF INVESTIGATION OF CONDITIONED AND UNCONDITIONED REFLEXES. N. KRASNOGORSKI, Jahrb. f. Kinderh. **122**:127 (Dec.) 1928.

Krasnogorski states that by means of the construction of a suitable vacuum chamber it has become possible to gather separately the saliva from the submaxillary gland. These glands react more rapidly and effectively than the parotid. Also, conditioned reflexes are more readily formed through the submaxillary gland.

Since it has become possible to study quantitatively the submaxillary secretions, a new method is available for the investigation of psychophysical relationships.

ABT, Chicago.

THE EXTERNAL INHIBITION OF CONDITIONED REFLEXES. A. JUSCHTSCHENKO, Jahrb. f. Kinderh. **122**:132 (Dec.) 1928.

According to Juschtschenko, artificially conditioned reflexes are inhibited by simultaneous foreign or strange stimuli. The effect of the latter is diminished by repetition. By such inhibition, the secretory reflexes (secretion of the parotid) are more readily affected than the motor (reflex of opening the mouth). Newly formed conditioned reflexes are more readily affected than the older reflexes and usually in the form of prolongation of the latent period. In smaller children, the inhibition of secretory and motor reflexes is more extensively developed. In such an investigation one should also consider the effect of earlier, unknown stimuli.

ABT, Chicago.

URANIN IN THE DIAGNOSIS OF MENINGITIS. J. C. SCHIPPERS and H. J. PETERS, Jahrb. f. Kinderh. **122**:202 (Dec.) 1928.

According to Schippers and Peters, uranin, an acid dye, may be successfully used in testing the permeability of the meninges. Since with sufficient concentration in the blood it also passes over into the cerebrospinal fluid, the optimal dose must be determined with which there is no transfer to the spinal fluid. For children this dose is 20 mg. per kilogram, by mouth. There is a detailed description of its quantitative determination in the liquor, blood, urine and feces. The uranin content of the blood rises rapidly. After fifteen minutes, there is considerable dye in the urine. In infants, there was not any greater permeability for uranin. In meningitis there is increased permeability; there is not, however, in meningismus.

In investigations of younger children by Walter's bromide method, there were permeability quotients in the region of the values for normal adults.

ABT, Chicago.

MYOTONIA CONGENITA. MAX BIELSCHOWSKY, J. f. Psychol. u. Neurol. **38**:199, 1929.

Bielschowsky had occasion to study five cases of this disease, three of which are described in detail in this contribution. All patients presented the characteristic symmetrical paralysis and flaccidity of the extremities, especially of the proximal segments. The tendon reflexes were either abolished or diminished, and the joints were hypotonic. The presence of "flail shoulders" and inability to hold up the head were common features. Some motion was possible with the hands and feet, but the excursions were unusually feeble. The hypotonic and paretic muscles were not atrophied, nor were they doughy in consistence. Of the auxiliary respiratory muscles, the intercostals and abdominal muscles were involved; aeration of the lungs was actually carried on by the diaphragm; it is therefore not surprising that all the children afflicted with the disease succumbed readily to acute respiratory infections. The condition was congenital in four cases, and in one it manifested itself during the third month of life, although in this patient's sister, also affected with the disease, the mother noticed the condition at birth. This factor is an important diagnostic point. In these two cases, the anatomic observations were the same as in the other three cases.

There was a striking uniformity in the microscopic observations in all cases. Slight deviations from the usual features were encountered in some of the cases, but these were of no significance as far as the symptomatology and nosology of the condition are concerned. Some of these abnormalities were: variations in the persistence of the external embryonal granular layer of the cerebellar cortex, heterotopy in some of the ganglion cells, smaller groups of ganglion cells in the cord, slight anomalies of the ependyma, polygyric type of convolution with cyto-architectonic abnormalities (cases 4 and 5), etc. While these anomalies per se are not of much importance, they are significant because they indicate a tendency to a dysgenesis which also has some bearing on the characteristic pathologic features of the disease.

The typical pathologic changes of myotonia congenita are found in the ventral horns of the cord, in the striated musculature and in the intramuscular innervation apparatus. In all cases the motor ganglion cells in the ventral horns, especially in the enlargements of the cord, were markedly diminished in number; this was more noticeable in the lumbosacral than in the cervical portion of the cord. In areas in which the motor cells were destroyed, there seemed to have been a peculiar predilection for individual groups of cells, in some areas for the median and in others for the lateral groups. These sites of predilection for cellular involvement were observed not only in different cases, but also in the same case. Most previous observers have reported similar observations. The involvement of the cells did not appear to be a purely systemic one; it affected not only motor cells, but the mesial cell groups, or the cells in Clarke's column. In case 3, the entire gray substance of the cord seemed to have been reduced although, even here, the loss of the multipolar ventral horn cells was by far the most prominent observation. The dorsal cord showed the least involvement. The so-called sympathetic cell groups in the lateral horns of the cervical and dorsal cords were almost entirely intact. The motor nuclei of the pons and medulla were only moderately affected. Quantitative involvement was observed in the facial nuclei in two cases (1 and 2), and the hypoglossal nuclei in cases 4 and 5 showed striking cellular defects. Every case showed qualitative variations in the motor nuclei of the hindbrain. The absence of clinical signs of bulbar involvement in these cases was striking. The nuclei of the ocular muscles were normal in every case.

There were no evidences of vascular or inflammatory processes to account for the cellular involvement, so that there could be no question that one was not dealing with a fetal poliomyelitis or some vascular disease as the basis of the cellular changes. Nor could any conclusions be drawn as to the nature and origin of the pathologic process from a study of the glial preparations. The cellular defects were replaced by fiber-forming astrocytes. The nestlike grouping of

astrocytes was, according to Bielschowsky, evidence that a large number of ganglion cells must have fallen out at one time and had soon been replaced by astrocytes.

A study of the ganglion cell in this disease raises the question whether one is dealing with a degeneration — a shrinking process — or with a maldevelopment. The discussion of this question, Bielschowsky says, "is as old as the disease itself," and has led to various theories as to its causation and as to whether or not it was identical with the spinal amyotrophies. He himself believes that the disease is a malformation or arrest of development during which the motor innervation of the striated muscles has remained inadequate quantitatively as well as qualitatively. The anatomic substratum is one of agenesis and dysplasia of the ventral horn cells with a consequent insufficient supply of axons for the muscles supplied by these cells and a failure of maturation of the axonal terminations. Bielschowsky attaches more importance to the lesions of the axons and their endings than to those of the ganglion cells. The essence of the pathologic process, according to him, is a defective muscular neurotization. With this conception as to the pathogenesis of the disease in mind, much of the speculation as to the reason for possible recovery can be dismissed; one can readily conceive that improvement may occur because later in life either some of the contact organs within the muscles become mature or motor fibers invade the immature contractile substance of the muscles, both of which processes have a tendency to enhance voluntary innervation of the defective muscles. Bielschowsky is certain that this gradual change in muscle neurotization is the only substratum for the increasing functional capacity noted in these cases during the period of improvement.

So many writers have attempted to put myotonia congenita in the same nosologic category as the Werdnig-Hoffmann type of muscular atrophy that the author feels it incumbent on himself to emphasize the points of differentiation between these two conditions. The changes in myotonia have the earmarks of arrested development, which is complete at birth. Werdnig-Hoffmann's atrophy is due to a regressive process in previously normal spinoperipheral neurons which leads clinically as well as pathologically to typical manifestations of secondary degeneration in the musculature. A relationship between the two is conceivable, but only causopathogenically; namely, the arrested development in myotonia and the progressive cell degeneration in Werdnig-Hoffmann's atrophy might be due to similar endogenous factors. Because in some cases of myotonia the neurologic symptoms are associated with obesity, genital hypoplasia and myxedematous skin changes, some observers (Glanzman) have attempted to attribute the disease to endocrinal disturbances, particularly of the hypophysis. These observations, however, are few, and the results of anatomic investigations in these cases from the endocrinal point of view do not support such a hypothesis.

It must be mentioned, however, that the histologic differential diagnosis may occasionally present great difficulties. This is especially the case when regressive processes are found implanted on the dysgenesis and hypoplasia of the ganglion cells. Even in the cases that are the subject of this communication, the vessel walls in the region of the ventral horns and the differentiated wide muscle fibers show some evidences of a progressive degeneration, but these are too insignificant and far overshadowed by the dysgenetic phenomena.

KESCHNER, New York.

THE POTASSIUM, CALCIUM AND MAGNESIUM CONTENTS OF CEREBROSPINAL FLUID. BELA EISLER, Ztschr. f. d. ges. exper. Med. **61**:549, 1928.

The potassium, calcium and magnesium contents in the cerebrospinal fluid and their relative ratios were determined for 154 patients. In the acute, purulent forms of meningitis (twenty-nine cases), little change was found in the amounts of potassium and calcium, but the amount of magnesium was increased several fold. In tuberculous meningitis (fifteen cases) there was an increase of potassium to from 15 to 20 per cent above the normal, and a diminution of calcium. No changes were found in twenty-eight cases of serous meningitis. There were no constant changes in thirty-eight cases of cerebrospinal syphilis. In two cases

of paresis there occurred an increase of potassium and magnesium, and a decrease of calcium after treatment with malarial parasites. In patients with tetany and spasmophilia, the calcium was particularly low. [ARCH. PATH.]

PSYCHOLOGIC TESTS IN VARIOUS TYPES OF CONGENITAL MENTAL DEFICIENCY. I. EISENSTEIN, Ztschr. f. Kinderforsch. **35**:563 (Aug. 1) 1929.

The attempt is made to classify mental deficiency on the basis of mental testing. Only thirty cases are described. REICHLE, Cleveland.

AN EXTRAORDINARY CASE OF DEFICIENCY IN READING ABILITY. L. ESSER, Ztschr. f. Kinderforsch. **35**:590 (Aug. 1) 1929.

A girl, aged 13 years, who showed an astonishing deficiency in reading ability but who was otherwise of average intelligence, was carefully studied and trained by the author. He believes that this defect was due to a peculiar abnormality of visual appreciation. By adapting the course of instruction to this difference in the child, he was able greatly to improve her reading ability.

REICHLE, Cleveland.

Diseases of the Eye

EMBOLUS OF THE CENTRAL RETINAL ARTERY IN A CHILD. VLASTIMIL KUBIN, Časop. lék. česk. **65**:1153 (July 30) 1926.

According to Leber, twelve cases of embolus of the central retinal artery have been reported in the literature. The author reports the case of an apparently normal girl, aged 10, who complained of a sudden loss of vision in the right eye. On examination, vision was lacking in the right eye and 10/10 in the left eye. The right pupil was larger and reacted slowly to light and in accommodation. Ophthalmologic examination showed a preserved rhomboidal islet between the macula and the papilla, with a background of practically normal coloration. The sepia-colored fovea centralis was surrounded by a light yellow, vertical rectangle, the temporal side of which was speckled with several lustrous white spots. This area was sharply defined by a white margin with incomplete terraced zones placed centrally. A red hemorrhagic spot was found in the upper nasal portion, and the periphery of the fundus was finely pigmented. STULIK, Chicago.

Diseases of the Skin

A CASE OF HYPERKERATOSIS PALMARIS ET PLANTARIS ASSOCIATED WITH AINHUM-LIKE CONSTRICTION OF THE FINGERS. J. E. M. WIGLEY, Brit. J. Dermat. **41**:188 (May) 1929.

A case is described in a girl, about 10 years of age, the only one of a family of four with any physical defect. Thickening of the palms and soles was first noticed at the age of 3 months, and the constricting bands around the fingers appeared at 7 years. Four photographs illustrating the distribution and character of the lesions accompany the article. Roentgenologic examination revealed a definite rarefaction of the bony phalanges. The process takes some months and is not accompanied by any degree of pain. The resemblance of the tapering of the fingers to that occurring in sclerodactylia and the lesions on the knuckles to localized scleroderma is commented on. WIEN, Chicago.

AN HEREDITARY ECTODERMAL DYSTROPHY. H. R. CLOUSTON, Canad. M. A. J. **21**:18, 1929.

One hundred nineteen cases of hereditary dystrophy of the nails and hair were analyzed. Forty of these were seen by the author. The patients were all of French-Canadian origin, and represented six generations. The condition of the

nail was a constant factor. The dystrophy followed the laws of mendelian hybrids; it was transmitted through both sexes and affected both sexes alike, and offspring with the dystrophy were dominant. Involved were the epidermis itself and its appendages, and there was clinical evidence that the teeth, the nervous system and those glands of internal secretion which are of ectodermal origin were also affected. The hair condition was a hypotrichosis from birth—a failure of the pilosebaceous system to develop. The dystrophy tended to diminish in succeeding generations, as to both incidence and severity, directly as living conditions were improved. The author estimates that there are more than 6,000 cases in America. The paper contains illustrative charts and photographs, and a critical review of the literature.

RATTNER, Chicago.

A CASE OF IDIOSYNCRASY RELATED TO EGG: RESEARCHES ON THE RÔLE OF ALLERGY AND IDIOSYNCRASY IN SOME DERMATOSES, ESPECIALLY ECZEMA. B. BLOCH and G. PRIETO, Ann. de dermat. et syph. **10**:461 (May) 1929.

The authors give an excellent summary of the present knowledge of allergy and idiosyncrasy. They report the case of an infant of 10 months with eczema in whom the ingestion of egg produced an erythematous-urticarial eruption and systemic symptoms. Contact skin tests with egg were positive, producing a macroscopic and histologic reaction akin to eczema. Passive transfer (Prausnitz-Kustner) tests were positive.

Proceeding from these observations, Bloch and Prieto discuss the biologic mechanism of idiosyncrasy. They regard it as a type of allergy (antigen-antibody reaction) characterized chiefly by being inherent and affecting only a few persons. They regard eczema as a typical idiosyncratic phenomenon. In their investigations of this case, they were able to provoke an eczematous reaction in the affected person, but not in the person passively sensitized, in whom an urticarial reaction occurred. Hence, they do not claim to have produced eczema by passive sensitization.

MICHAEL, Houston, Texas.

CONTRIBUTION TO THE QUESTION OF HERPES ZOSTER. S. A. GLAUBERSOHN and R. A. VILLFAND, Ann. de dermat. et syph. **10**:609 (June) 1929.

In this study, inoculation of zona from child to child by means of skin scarification was attempted. Successful inoculations and reinoculations were obtained with recent vesicular contents. Failure attended the use of spinal fluid of patients with zoster. The authors conclude that this disease is infectious and that the skin is the portal of entry.

MICHAEL, Houston, Texas.

EXPERIMENTAL INVESTIGATIONS ON ALLERGY AND IMMUNITY IN TRICHOPHYTON INFECTIONS. E. RIVALIER, Ann. de dermat. et syph. **10**:618 (June) 1929.

Rivalier reviewed previous work on these subjects and added personal investigations of *Trichophyton* infections in guinea-pigs. He found that trichophytic infection in these animals, when produced by a highly pathogenic organism, produced profound tegumental and systemic alterations of an allergic type. Immunity, in the strict sense, does not seem to be present. Cutaneous manifestations of allergy are sufficient explanation of the response of an animal to a second attempt at reinoculation, and there is perfect accord between the degree of resistance to a new infection and the intensity of allergy. The greater the sensibility the more marked the cutaneous reaction to infection, and vice versa.

The author mentions the loose use of the term immunity, and so far as *Trichophyton* infection is concerned, believes that allergy is the paramount mode of defense.

From the practical standpoint, it becomes evident that preventive or curative treatment with *Trichophyton* is fruitless. If it has any effect, it would produce a diminution of allergy and therefore a lowered resistance to infection.

MICHAEL, Houston, Texas.

FAMILIAL SCLERODERMA. LOUSTE, JUSTER and MICHELET, Bull. Soc. franç. de dermat. et syph. **36**:440, 1929.

An instance of scleroderma in mother and son is reported. The question of syphilis or other common infection is raised.

MICHAEL, Houston, Texas.

TREATMENT OF TINEA CAPITIS BY THALLIUM ACETATE EPILATION. L. M. PAUTRIER and WORINGER, Bull. Soc. franç. de dermat. et syph. **36**:491, 1929.

Employing doses of 8 mg. per kilogram of body weight, the authors obtained eight cures in ten cases. The failures were apparently due to an early regrowth of the hair. No severe untoward effects were produced by the remedy.

MICHAEL, Houston, Texas.

ECZEMA MARGINATUM IN A NURSLING. L. PERIN, Bull. Soc. franç. de dermat. et syph. **36**:580, 1929.

The eruption, typical of this disease, began during the third week of life. Scale preparations were positive for fungi.

MICHAEL, Houston, Texas.

DISSEMINATED TUBERCULOSIS VERRUCOSA CUTIS FOLLOWING MEASLES. A. SEZARY and POUMAILLOUX, Bull. Soc. franç. de dermat. et syph. **36**:597, 1929.

A patient, aged 15, exhibited numerous lesions of verrucous tuberculosis, which had appeared in successive crops following uncomplicated measles. On the skin typical patches of lupus vulgaris were also present. The original focus of the infection could not be determined.

MICHAEL, Houston, Texas.

Miscellaneous

STATISTICAL REPORT OF INFANT MORTALITY FOR 1928. American Child Health Association, August, 1929.

This is the annual compilation of infant mortality rates in 729 cities of the United States for the year 1928. The death rate among infants was 68.3 for the 719 cities of the birth registration area. This is an increase over the 1927 figure of 64.9. These rates represent the number of deaths among children under 1 year of age for each 1,000 live births.

SANFORD, Chicago.

SICKLE-CELL ANEMIA WITH CASE REPORT OF SPLENECTOMY. JOHN F. LANDON and A. VICTOR LYMAN, Am. J. M. Sc. **178**:223 (Aug.) 1929.

The author reports a case of sickle cell anemia in which splenectomy was performed. A colored child, aged 4 years, was under observation for five months with a provisional diagnosis of von Jaksch's anemia, Gaucher's disease also being considered. At the end of this time, splenectomy was performed. A study of the spleen was made by various pathologists, and a diagnosis was finally made by Dr. Martha Wollstein who reported finding sickle-shaped red cells in the section of the spleen. Following this report, blood smears were made and the sickle-shaped cells were found in the blood.

Following the splenectomy the patient was observed for fourteen months. The general condition showed a marked improvement but persistence of the sickle-shaped cell in the blood.

The author reviews the cases of splenectomy and draws attention to the fact that in no case was the presence of sickle-shaped cells abolished by splenectomy.

HENSKE, Omaha.

THE OUTCOME OF 625 PREGNANCIES IN WOMEN SUBJECTED TO PELVIC RADIUM OR ROENTGEN IRRADIATION. DOUGLAS P. MURPHY, Am. J. Obst. & Gynec. **18**:179 (Aug.) 1929.

The author made a study of the outcome of pregnancy in 625 women who had been subjected to irradiation in therapeutic doses before or during pregnancy.

Twenty-four per cent of pregnancies, or a number somewhat less than usual, ended in abortion. This percentage was greatly increased, however, if the irradiation was given after pregnancy had occurred.

Seventy-six, or 12 per cent, of the children born were unhealthy; thirty-eight of these children were born of mothers who had received irradiation before conception. This would correspond to statistical percentages for fetal deaths in patients who had not been subjected to irradiation. Irradiation after conception did not tend to increase the rate of stillbirths or deaths in the first year of life. It markedly increased, however, the percentage of fetal deformities; one third of these children gave evidence of gross deformity, particularly mental. A large number were microcephalic in type. Seventeen cases of this group revealed that prenatal irradition is an extremely dangerous procedure. The author recommends routine pre-irradiation curettage or immediate termination of pregnancy during which irradiation has been given.

ALLEN, Chicago.

SERVING THE CHILD IN FARGO. PART THREE OF THE FINAL REPORT OF THE FARGO DEMONSTRATION. BULLETIN 9. Commonwealth Fund Division of Publications, 1928.

The central purpose of the five-year child health demonstration in Fargo, N. D., was to show how certain activities designed to better the health of children might be built up as an integral part of a balanced and practicable public health scheme for a small city. In this report the activities are described in sufficient detail to give other cities, which might wish to undertake such a program, the essential facts to work on. The details of the health program are described under three groups dealing with: infants and preschool children, school children and prenatal and postnatal maternity service. In the preschool period the preliminary and most essential step was the establishment of contact by the nurse with families who had young children, in order to interest the parents in adequate health supervision, whether by the family physician or by the demonstration health center. A high degree of cooperative relationship was developed between the demonstration clinic and the medical society, and through the latter and the individual physician the health center undertook no treatment and only examined such children as had no family physician — the purpose of the demonstration being to interest both the parents and the medical profession in developing an adequate supervision of the health of preschool children and to function, not as a foreign body in the community, but as an integral part of the health activities already existing. Of 2,311 preschool children examined by the health centers, 663 were found to have defects sufficiently serious to require medical attention. The most frequent defects in both sexes were dental and nasopharyngeal conditions. Boys showed a large number of genital defects.

Similarly, among the school children the demonstration tried to develop a high degree of coordinated interest between the children, the parents, the teachers and the school nurse. Health topics were discussed by the teacher and class — the teacher encouraged the children to keep their own health records, and similar records were kept by the parent. There was a daily inspection of hands, teeth, etc., and those children who showed defects were referred to the school nurse. Among school children, dental, nutritional and nasopharyngeal defects were the most common.

The demonstration had three major objectives: (1) to improve the health status of Fargo children; (2) to convince the Fargo public that children health service was worth continuing at local expense, and (3) to make various professional groups more aware of their opportunities to build up and conserve the health of children.

The second objective has been attained in a large measure. It is difficult to measure the exact degree of accomplishment of the first aim, but there is no doubt in the minds of those who have watched Fargo children in the last five years that their health has improved. Infant mortality rates — high in comparison with those of the Registration Area before the demonstration — are now relatively low.

The results of the third aim are still more difficult to measure, but not only has the cooperation between all professional groups been increased, but the citizens of Fargo themselves seem to show a sense of responsibility for the work being carried on, far greater than citizens ordinarily exhibit for the health work of their communities.

This mimeograph is an interesting record of the manner in which cooperation may be developed among various existing professional groups by an outside agency, the basic plan of which is to work in cooperation with them and utilize their resources to the fullest extent.

PEARSON, Philadelphia.

VACCINE AND SERUM THERAPY IN PEDIATRICS. W. MCKIBBEN, J. Florida M. A. **16**:117 (June) 1929.

The author presents a comprehensive summary of the present status of vaccines and serums as applied to the prevention and treatment of seventeen disorders and diseases of childhood. The value of these agents as preventive and curative measures is discussed from the author's point of view. He includes the generally accepted opinions of leading authorities on this subject.

The disorders dealt with are common colds, diphtheria, epidemic encephalitis, erysipelas, infantile paralysis, influenza, measles, meningitis, pneumonia, rabies, rheumatic fever, scarlet fever, smallpox, tetanus, tuberculosis, typhoid fever and whooping cough. The paper is sufficiently comprehensive to enable the author to present in detail most of the salient features pertaining to vaccine and serum prophylaxis, and treatment for the seventeen disorders considered.

LOVE, Jacksonville, Ill.

OBSERVATIONS ON THE NATURAL HISTORY OF ASTHMA IN CHILDHOOD. STEVENS S. BULLEN, New York State J. Med. **29**:545 (May 1) 1929.

The records of 235 patients who developed asthma under 15 years of age and 265 who had the first attack after 15 years are compared; 80 per cent of the younger group give a family history of asthma and 10.6 per cent had had eczema. No racial peculiarity was found. Pulmonary tuberculosis was infrequent. The initial colds were usually allergic coryza. Sinusitis was found in but 13 of the children. A larger number of the 500 had the first asthmatic attack in the first five years of life. The results of the treatment are more satisfactory with patients who develop asthma in childhood.

AIKMAN, Rochester, N. Y.

INFECTIONS AS A SOURCE OF RECURRENT EPILEPTIFORM SEIZURES IN CHILDREN. DONALD PATERSON, Arch. Dis. Childhood **4**:138 (June) 1929.

Twelve cases of convulsions with acetonuria are reported. There is no indication as to whether the acetonuria preceded or followed the convulsions. Contrary to a large amount of scientific evidence that has been published in the United States, it is suggested here that acetone or some accompanying chemical product might be the cause of the convulsions.

PETERMAN, Milwaukee.

THE INTELLIGENCE OF EPILEPTIC CHILDREN. SHEPARD DAWSON and J. C. M. CONN, Arch. Dis. Childhood **4**:142 (June) 1929.

Forty-nine epileptic children were given mental tests, and a study of the intelligence was made. The intelligence ratio, the mean of the whole group, was 80.65 per cent. This was below the average of the 991 other children in the hospital, who had a mean of 90.57 per cent. The intelligence ranges from feeblemindedness to 117 per cent. The intelligence ratios of the brothers and sisters of

the epileptic children (twenty) was 91.20 per cent. There is no statement of the type of epilepsy in these children, nor is there any evidence of the duration of the diseases. Both factors are of considerable importance in making an interpretation of the intelligence, and should be considered before the conclusions are drawn that the average intelligence of epileptic children is below normal.

PETERMAN, Milwaukee.

THE EFFECT OF VARIATION OF THE DIET IN RHEUMATIC CHILDREN. A. R. THOMSON and S. H. EDGAR, Arch. Dis. Childhood **4**:152 (June) 1929.

Twenty-two children convalescing from rheumatism, most of them with heart lesions, were divided into two equal groups. One was given a diet high in protein, the other a diet high in carbohydrate. Estimations of the blood p_H, carbon dioxide, urea, nonprotein nitrogen and uric acid were made before and after five months of diet. There was no appreciable difference between the two groups in respect to the chemical observations or the clinical progress of the disease.

PETERMAN, Milwaukee.

ABDOMINAL PAIN IN CHILDREN. H. W. CARSON, Canad. M. A. J. **20**:587 (June) 1929.

The author describes briefly the distribution of the nerves of the abdominal viscera, and the relations of the visceral and somatic spinal centers.

In regard to the inflammatory condition, appendicitis is the outstanding disease; 50 per cent of the author's cases of acute appendicitis occurred in children under 12 years of age.

It is questionable whether chronic appendicitis is ever present in children.

Recurring abdominal pains sometimes occur as the result of hydrops of the appendix or of thread worms.

The author emphasizes the fact that local signs are often misleading in an infant.

Acute tuberculous peritonitis and acute pneumococcal peritonitis are briefly discussed. The author believes that the latter is becoming more common.

Intestinal obstruction has such a definite picture that it should be recognized and an operation performed immediately. Interesting statistics are given showing the marked improvement in prognosis in recent years.

From 1893 to 1895, at the London Hospital, fifty-five patients were treated, with a mortality of 82 per cent. Today, recovery is the rule; the author's last seventeen patients with this condition all recovered.

Chronic abdominal pain is discussed briefly. The author states his belief that enlarged tuberculous glands in the mesentery are a common cause of chronic abdominal pain.

MOORE, Omaha.

ABNORMAL DENTITION. JEAN SMITH, Proc. Roy. Soc. Med. **22**:643 (March) 1929.

A patient, aged 6 years, showed on examination two canine teeth occupying the position of upper central incisors, four temporary first molars and four permanent first molars posterior to the deciduous ones. The hair of the patient was fine, fair and sparse. The intelligence was normal. Dental films showed no unerupted teeth. Roentgenograms of the epiphyses and of the sella turcica showed a normal condition. No teeth were cut until the eighteenth month; these were the canines in the position of the central incisors. The four temporary first molars were cut between the second and third years, and the four permanent molars at about 5½ years.

WILLIAMSON, New Orleans.

SUDDEN DISLOCATION OF THE HIP IN A PARALYZED CHILD, ON THE NONPARALYZED SIDE, CAUSED BY EXTREME ADDUCTION. M. ROEDERER, Bull. Soc. de pédiat. de Paris **27**:21 (Jan.) 1929.

A female idiot, aged 13, was well developed and well nourished, but had paresis of the left leg. After sleeping in a berth on the train, she was unable to stand up. Roentgenologic examination showed dislocation of the right hip. In the absence

of signs of arthritis, the dislocation was thought to be caused by the extreme adduction of the thigh, which the patient used as a support. The author points out that a dislocation of the hip on the paralyzed side has been repeatedly described in the literature, and that it occurs as a result of the atrophy of the gluteal muscles, but that a dislocation of the normal hip in a child with one-sided paralysis is exceptional.

BENJAMIN, Montreal, Canada.

THE INFLUENCE OF EMOTIONAL SHOCK AND THE RÔLE OF THE NERVOUS SYSTEM IN THE ACIDOSIS OF CYCLIC VOMITING. M. DESHAYES, Bull. Soc. de pédiat. de Paris **27**:22 (Jan.) 1929.

The author cites cases of cyclic vomiting in which the onset of attacks of vomiting followed emotional stimuli. He states his belief that emotional shock is the chief cause of onset. The acidosis is due to a disturbance of the ketogenic function of the liver which in some way depends on vagus hyperexcitability, which he thinks is present in all these patients. Marfan agrees that emotional excitement is a well known factor in the causation of some cases of cyclic vomiting, but he believes that any sudden change, such as the onset of an acute infection, may cause vomiting. He thinks that faulty liver function is probably a cause of the accumulation of ketone bodies in the blood, but he makes the suggestion that because of the antiketogenic action of insulin a disturbance of the pancreas may have something to do with the onset of vomiting.

BENJAMIN, Montreal, Canada.

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS: ANATOMIC EXAMINATION OF THE PYLORUS THREE MONTHS AFTER CURE BY EXTRAMUCOUS PYLOROTOMY. E. LESNE, P. FREDET and M. COFFIN, Bull. Soc. de pédiat. de Paris **27**:31 (Jan.) 1929.

A breast-fed boy, who weighed 3,080 Gm. at birth, weighed 3,000 Gm. at 30 days of age. At 31 days he began to have projectile vomiting immediately after feedings. He was first seen by the authors at the age of 6 weeks, when his weight was 2,500 Gm. He showed visible gastric peristaltic waves, and some small reversed peristaltic waves, but no palpable tumor. The mother first noticed the peristaltic waves when he was 11 days of age. In the hospital, roentgenologic examination showed pyloric obstruction; this was further confirmed by the fact that the infant vomited 70 Gm. after taking only 26 Gm. at each of his feedings, seven and four hours previously. It took some time to get the consent of the parents to operate on the infant. The operation was done at the age of 7 weeks, after the child had lost 26.4 per cent of his birth weight. He made an uninterrupted recovery and began to gain at the rate of 30 Gm. per day. Three months later, he died of bronchopneumonia.

At necropsy, the stomach was normal; there were no adhesions and no trace of the operative incision on the pylorous; Fredet could feel a slight thickening for about 2 cm. on the pylorus, where he had cut it, but the consistency of this was not at all like that of the original olive-shaped tumor. Photographs of sections under low power and high power magnifications showed that there was practically complete restitution to normal. This agrees with the observations of Rachford and Martha Wollstein and adds to the favorable consideration that the Fredet operation already enjoys.

BENJAMIN, Montreal, Canada.

BLOOD TRANSFUSION AND VACCINE THERAPY IN DISEASES OF CHILDREN. C. BARROSO, Arch. de pediat., Rio de Janeiro **2**:338 (July) 1929.

Barroso gives from 50 to 200 cc. of blood, depending on the size of the child, and an autogenous vaccine as soon as suitable cultures can be made. This is repeated at forty-eight hour intervals as needed. This treatment is successful in all cases in which there is a specific organism involved. Cases that were of particular interest were those of pyelocystitis, lobar pneumonia and staphylococcus septicemia.

SANFORD, Chicago.

INTRAPERITONEAL TRANSFUSION. A. CASTELLANOS, Bol. Soc. Cubana de pediat., Havana **1**:432 (July) 1929.

Castellanos reviews the literature and concludes that intraperitoneal transfusion of blood is indicated in all cases in which it is not especially desirable to have a rapid absorption. This refers particularly to anemia, wasting diseases, infections and postoperative cases. This method is easy and safe in the young infant, in whom the minuteness and extra-collapsibility of the veins would make intravenous transfusion of great difficulty.

SANFORD, Chicago.

THE PROTECTION OF INFANTS IN MONTEVIDEO. ISMAEL VALDES VALDES, Bol. Inst. internac. Am. de Protec. Inf. **2**:414 (April) 1929.

The children of Montevideo are supervised by two branches of the National Board of Public Health, the Department for the Protection of Infants taking charge of all children under 3 years of age and the Damaso Larranago Home caring for all other children.

In an infant home, children are cared for whose parents are incapable of caring for them. Day nurseries supplement home care. In 1928, 1,735 children were cared for in foster homes. A wetnurse department was conducted. All legal wards receive periodic medical examination. To provide medical inspection and assistance to children of destitute families, dispensaries exist which in addition to medical care act as distributing centers for milk and milk foods. Every day a central milk kitchen prepares the milk products that are required at the dispensaries. Canteens provide food for nursing mothers. The department also has its own laboratory. The expense of the department, including nine infant dispensaries, is about $1,000.00 a day. Three private institutions cooperate in the work of the department.

LESLIE, Evanston, Ill.

SCHOOL ANTHROPOMETRY. R. SCHIAFFINO, Bol. Inst. internac. Am. de Protec. Inf., April, 1929, vol. 2, no. 4.

The author presents anthropometric data from measurements of the school children of Montevideo. He finds that up to the age of 14, the children are taller than those described in any other statistical research. After the age of 14, the influence of race origin becomes noticeable. The height-weight-chest proportion is regular and shows a uniform development of all the body. The type of growth is peculiar in its uniformly ascending character, with a gradual yearly increase in the periods of greater growth, but never shows the sudden ascents that are noticed in children of other countries.

LESLIE, Evanston, Ill.

SIX MONTHS OF SCHOOL HYGIENE. OSCAR CLARK, Bol. Inst. internac. Am. de Protec. Inf. **3**:38 (July) 1929.

The author, who is chief inspector of the school medical service of Rio de Janeiro, reports on the duties and activities of the school physicians and nurses. This entails the usual supervision records of examination and instruction. The author demands the creation of school clinics which may become centers for the treatment of destitute children, and real health centers for the community.

LESLIE, Evanston, Ill.

THE SCHOOL MEDICAL SERVICE IN CHILE. LUIS CALVO MCKENNA, Bol. Inst. internac. Am. de Protec. Inf. **3**:73 (July) 1929.

The school medical service of Chile is undergoing a severe reorganization. The School Health Institute, created in the beginning of 1928, was dissolved by the government at the end of the year; the medical service was then committed to the care of the Ministry of Hygiene and Social Welfare. This offers the advantage of carrying the mentioned service to all parts of the country where

there is a sanitary physician; that is, to about 100 localities. Children will be treated in the hospitals, Red Cross institutions or other specified establishments, or by the family physician. In all cases the sanitary nurse supervises the treatment.

LESLIE, Evanston, Ill.

SCHOOL MEDICAL ORGANIZATION IN URUGUAY. RAFAEL SCHIAFFINO, Bol. Inst. internac. Am. de Protec. Inf. **3**:99 (July) 1929.

There was considerable expansion in the work of the school medical board in 1928. Thirteen medical inspectors, three specialists and twelve visiting nurses supervise the health of the school population of Montevideo. Specialized clinics are in operation and also welfare work comprising open air schools, nutrition work, classes for backward children and summer camps. The rest of Uruguay, with a population three times as great, has no school medical service.

LESLIE, Evanston, Ill.

THE EVOLUTION OF THE SCHOOL MEDICAL SERVICE IN BUENOS AIRES IN RECENT YEARS. ENRIQUE M. OLIVIERI, Bol. Inst. internac. Am. de Protec. Inf., July, 1929, vol. 3, no. 1.

The School Medical Service of Buenos Aires was founded in 1886, but until 1924 lacked adequate premises and materials. Since then, it has obtained large headquarters and has enlarged its staff to thirty-eight physicians, four dental inspectors, sixteen assistant dentists, two assistant chemists, seventy-five hygiene visitors and one specialist in mental defects. Adequate laboratories and clinics are available, and the latter are well attended. Six open air schools, and mountain and seaside colonies are also operated. Six canteens are in existence.

LESLIE, Evanston, Ill.

STRANGULATED HERNIA IN EARLY INFANCY. MARTINEZ VARGAS, Med. de los Niños **30**:136 (May) 1929.

In an article appearing in two issues of this journal, the subject of hernia in infants is treated exhaustively. The author has seen three cases of tuberculosis of the hernial sac. Vomiting of fecal matter, abdominal distention, pallor and sweating are common when the hernia becomes strangulated. Attempts at manual reduction are made after the patient has relaxed in a warm bath for twenty minutes, the temperature of the water being 36 C. The hips are then elevated, and gentle pressure is applied to the tumor. The cooling effect of a compress of ether on the mass reduces the congestion and facilitates reduction. A light chloroform anesthetic may be tried if the bath is not effective. An operation which has been successful is described. After operation, nothing but water is given for thirty-six hours.

DWYER, Kansas City, Mo.

TWELFTH CONGRESS OF THE SPANISH ASSOCIATION FOR THE PROGRESS OF SCIENCE. MARTINEZ VARGAS, Med. de los Niños **30**:144 (May) 1929.

With King Alfonso presiding, the Spanish and Portuguese associations met at the University of Barcelona. Dr. Vargas addressed the section on scientific medicine on neoplasm in infancy, diphtheritic paralysis and prolapse of the rectum. It was proposed that there be an interchange of advanced students between the medical schools of Barcelona and Cordoba, Argentina.

DWYER, Kansas City, Mo.

Society Transactions

CENTRAL STATES PEDIATRIC SOCIETY

Fourteenth Annual Meeting, Sept. 27 and 28, 1929, Milwaukee

J. GURNEY TAYLOR, M.D., *President*

A. H. PARMELEE, M.D., *Secretary*

Program for Friday Morning, Sept. 27, 1929

Rubella Immediately Following Rubeola; Case Report.—Dr. J. Gurney Taylor.
The Development of Bone.—Dr. E. J. Carey.
Urticaria Pigmentosa.—Dr. G. H. Fellman.
Suprarenal Hemorrhage.—Dr. A. L. Kastner.
Chemical Blood Analysis and Its Importance to the Hospital.—Dr. J. C. Bock.
Staphylococcic Empyema in an Infant, Aged Two Months; Recovery.—Dr. R. M. Greenthal.
The Use of Toxoid for Immunization Against Diphtheria.—Dr. A. B. Schwartz.
Use of Toxoid for Immunization Against Scarlet Fever.—Dr. H. O. McMahon.
Nutritional Requirements of Sick Children.—Dr. M. G. Peterman.
Transfusions in Children.—Dr. S. J. Seeger.
Juvenile Flatfoot.—Dr. H. C. Schumm.
Malignant Hypertension.—Dr. F. D. Murphy.
Osteomyelitis; with Fistula of Bladder; Case Report.—Dr. N. W. Bourne.
Chondrodysplasia.—Dr. W. P. Blount.
Cerebral Injury at Birth with Maldevelopment and Myxedema.—Dr. R. L. Ritchie.
Intussusception with Rupture and Peritonitis.—Dr. John Doctor.

RUBELLA IMMEDIATELY FOLLOWING RUBEOLA. DR. J. GURNEY TAYLOR.

Rubella has frequently been reported as occurring in children convalescing from scarlet fever, but I have found little reference in the literature relative to it immediately following measles.

It is true that relapses commonly occur in measles, and therefore this sequence requires the ruling out of such a possibility.

Schamberg and Kolmer (Acute Infectious Diseases, 1928) cite a number of observers who have reported relapses occurring in from two to four weeks after the first attack.

A boy, aged 4 years, developed well defined measles, with definite Koplik's spots preceding the onset of eruption, as well as all the other classic symptoms. He had apparently entirely recovered from this.

In the third week after the onset of the measles he was exposed to a child with rubella; on the eighth day following the exposure he developed a mild, well defined maculopapular eruption and marked adenopathy, especially involving the postauricular and cervical glands. There was almost no prodromal disturbance, and no desquamation. A diagnosis of rubella was made.

The case is of interest first, because of a definite exposure to rubella at the end of convalescence from the measles and secondly, on account of the short period of incubation.

The period of incubation is commonly regarded as of longer duration than that of measles.

Griffith considered this variability of time of incubation as one of the diagnostic characteristics of rubella, stating that "in measles we find a fixed period of incubation."

Forcheimer's table (Rubella in Twentieth Century Practice of Medicine) which has been added to by Schamberg (Acute Infectious Diseases, 1928, p. 557), includes many variable observations as noted by clinicians, in which they give the range of incubation as from four to twenty-one days.

THE DEVELOPMENT OF BONE. DR. E. J. CAREY.

The paper as well as the exhibit covered the pathogenesis of rickets, achondroplasia and myositis ossificans, by means of plastine plates, photomicrographs and microscopic slides. Experimental evidence of the influence of the musculature on the direction of normal and abnormal bone growth by localized muscular excisions at the knee joint, pectoralis major of the chest and temporalis of the skull in growing puppies was shown; an amyotonic decompression of the skeleton was produced comparable to the rachitis-epiphyses, rosary and parietal bosses of the skull. Experimental evidence of myogenic dwarfism (achondroplasia) was demonstrated by extensive muscle work of puppies in a motor driven treadmill. Mounted dogs were presented as experimental results. The blood supply of epiphyseal bone in relationship to the pressure of joint mobilization was demonstrated.

URTICARIA PIGMENTOSA. DR. G. H. FELLMAN.

A girl, aged 10 months, had been breast fed since birth and was on a diet suitable for her age. The father and mother were living and well. The infant was well; when she was 2 months old, an eruption appeared on the trunk which consisted of elevated macular areas on which a nodular or vesicular lesion appeared. The eruption was accompanied by pruritus and when the acute symptoms subsided left brownish pigmented areas such as were visible at the time of examination.

Ormsby (Skin Diseases, 1927) classified the lesions of urticaria pigmentosa as (1) plain macular lesions; (2) nodular types and (3) mixed varieties of both types. The lesions vary in color through all shades of brown, yellowish brown, and reddish brown and become bright red if irritated. The lesions are usually discrete but may coalesce and vary in size from that of a split pea to that of a coin.

Fraser, in a recent article, stated that urticaria pigmentosa is a rare disease, less than 350 cases having been reported, and that it usually begins in infancy and continues to puberty. Sutton's figures show that about 46 per cent of the cases occur in the first year and 67 per cent before the thirteenth year.

The cause of these lesions of the skin is unknown; sometimes, shock to the nervous system has seemed to be the causative factor. The cause has been attributed to insect bites. Little stated that there is an overproduction of the mast cells of the skin and attributed it to a congenital disorder of the blood of the same class as hemophilia. Microscopically, the lesions show infiltration of mast cells into the corium and an increase of the pigment in the basal layer of the rete.

Treatment consists of the use of antipruritic lotions and ointments for relief of the symptoms, but these do not cause the disappearance of the lesions. Some have suggested roentgen treatment but this, also, lacks confirmation.

SUPRARENAL HEMORRHAGE. DR. A. L. KASTNER.

Suprarenal hemorrhage, except certain types, is not a rare condition, and the incidence of this observation at necropsy was given. A short tabulation of seven cases was shown, and one case of suprarenal apoplexy was reported. Some phases of the condition were illustrated with lantern slides. The symptoms, diagnosis and treatment of suprarenal hemorrhage were briefly discussed.

CHEMICAL BLOOD ANALYSIS AND ITS IMPORTANCE TO THE HOSPITAL. DR. J. C. BOCK.

The paper mentions the fundamental principles of the various colorimetric methods in use in this country, especially the system of blood analysis by Folin

and Wu. The methods of Bang as used in the analysis of blood are discussed. Serum analysis by refractometry and its clinical application are emphasized.

There follows a discussion of the problems confronting the hospital laboratory as far as the chemicodiagnostic work is concerned. The problems of the technician's competence and the supervision of the chemical laboratory are critically considered. The attitude of the clinician toward the laboratory is discussed.

STAPHYLOCOCCIC EMPYEMA IN AN INFANT, AGED TWO MONTHS; RECOVERY. DR. R. M. GREENTHAL.

P. G., aged 2 months, contracted an acute infection of the upper respiratory tract on Dec. 15, 1928. Later, pneumonia of the upper lobe of the left lung developed. On December 28, empyema was diagnosed. The infant was taken to the hospital and placed in an oxygen chamber containing 60 per cent of oxygen. Aspiration of the chest on the left side revealed pus which on culture gave a pure growth of *Staphylococcus aureus*. A blood transfusion was then given. On January 1, a rib resection was performed while the infant was in the oxygen chamber. A few hours later, a second transfusion was given. The temperature became normal six days after operation, and recovery was complete.

The prognosis in young infants is always grave. A few recoveries have been reported in patients with pneumococcus infection. In a search of the literature I found no case similar to this one. A blood transfusion before and after operation, the use of an oxygen chamber and a good previous state of nutrition of the infant appeared to be the deciding factors for recovery.

THE USE OF TOXOID FOR IMMUNIZATION AGAINST DIPHTHERIA. DR. A. B. SCHWARTZ.

In a series of 128 children given toxoid prepared by the Connaught Laboratories, University of Toronto, 98 per cent showed a negative reaction to the Schick test from three to six months after the injection of three doses. Only one local reaction was encountered. This occurred in an infant sensitive to veal broth.

Schick tests on this group were done with material prepared by the New York State Board of Health Laboratories. Standard Schick syringes were used. Tests were all done by the same person and read on the fifth day.

Conclusions.—1. In the preschool child, toxoid obtained from reliable sources offers the ideal method of diphtheria immunization. (*a*) It gives a greater percentage of immunity than toxin-antitoxin. (*b*) It avoids serum sensitization. (*c*) It provokes no reaction.

2. Any program of diphtheria immunization should include proper Schick testing.

3. The best age for diphtheria immunization with toxoid is about 1 year.

USE OF TOXOID FOR IMMUNIZATION AGAINST SCARLET FEVER. DR. H. O. MCMAHON.

Dr. McMahon reviewed various methods which have been used to produce immunization to scarlet fever. The use of the filtrate of the streptococci cultures by the Dicks instead of the vaccine as first proposed by Gabretschewsky had a distinct advantage in that in proper dilutions the filtrate could be used in a skin test for susceptibility. The recent tendency is to give large immunizing doses. McMahon used a toxoid prepared by Dr. Donald Fraser of the Connaught Laboratories of Toronto and gave it to fifty-five children showing positive reactions to the Dick test at three week intervals in doses of 0.5, 1.0 and 1.5 cc. The injections were started on April 15, and by August 15, 72 per cent of the children showed negative reactions to the Dick test. There was only one even moderately severe reaction, and the fact that immunization was accomplished by means of three injections rather than the customary five gives the toxoid a definite advantage.

NUTRITIONAL REQUIREMENTS OF SICK CHILDREN. DR. M. G. PETERMAN.

A study was made of the nutrition of fourteen children in the hospital with chronic disease of the bone, all but five of whom were tuberculous. Observations were made of the food requirements, appetite, choice of foods selected, the influence of vitamin B and the effect of supplementary foods, especially malted milk. The age, sex, height, weight, normal height and weight and surface area were determined and the total calories, carbohydrates, protein, and fat ingested. The mineral salts and vitamins were provided. Physical examinations, blood counts, and urinalysis were made at monthly intervals or oftener. The body height or length and weights were taken weekly. Only three patients made expected gains in weight; seven made slight gains; one held his weight, and three patients lost weight.

TRANSFUSIONS IN CHILDREN. DR. S. J. SEEGER.

This paper is based on a carefully studied series of 267 transfusions done in the past year at the Milwaukee Children's Hospital. One hundred and ninety-six were done by the multiple syringe or so-called syringe cannula method, 31 with the paraffin tube, and 40 by the citrate method. There were three reactions in this series, two following transfusion by the multiple syringe method and one following transfusion by the paraffin tube method. In all of the 267 cases the blood was given intravenously either in the forearm, the leg or the external jugular vein. The longitudinal sinus was not used.

The importance of proper hospital organization for the doing of transfusions is stressed. The most gratifying results will follow the assignment of this work to one member of the hospital staff.

The importance of special cross-matching as well as regular matching and grouping is emphasized by the fact that in sixty instances donors found to be in the proper group revealed no incompatibility by the so-called regular matching, while special cross-matching of patients' serum and donors' corpuscles or donors' serum and patients' corpuscles showed agglutination.

The various arguments for and against citrate in transfusions are discussed and experimental work confirming Gichner's observations reported.

This series of transfusions was given for the following conditions: secondary anemia (in forty cases), staphylococcic septicemia (in eight cases), streptococcic septicemia (in three), infectious diarrhea (in fifty-nine), pneumonia (in thirty-nine), von Jaksch's anemia (in three), marasmus, scurvy, rickets, endocarditis, hemophilia, prematurity, burns, cerebral hemorrhage, pulmonary tuberculosis, septic arthritis, peritonitis, meningitis, hypertrophic pyloric stenosis, malnutrition, mastoiditis, encephalitis, Pott's disease, hemorrhagic nephritis, enteritis, postoperative hemorrhage, empyema, erysipelas, furunculosis, scarlet fever, pyelitis and urticaria.

A plea is made for a general extension of the indications for transfusion. The operation should not be regarded as a remedy for hemorrhage, shock or profound anemia alone.

JUVENILE FLATFOOT. DR. H. C. SCHUMM.

This article appears in full in this issue, p. 1273.

MALIGNANT HYPERTENSION. DR. F. D. MURPHY.

A case of malignant hypertension occurring in a girl, aged 10 years, was studied at the Milwaukee Children's Hospital for a period of two years. Clinically, the persistent hypertension, the typical retinitis, the progressive downward course with an adequate renal function fulfilled the requirements for a diagnosis of malignant hypertension. Death occurred on May 27, 1929, and post mortem examination furnished morphologic evidence to confirm the clinical diagnosis. Lantern slides demonstrated the histologic changes in the small arteries and arterioles of various structures of the body.

OSTEOMYELITIS WITH FISTULA OF THE BLADDER. DR. N. W. BOURNE.

A boy, aged 13 years, who had been treated for two years for multiple osteomyelitis, complained of terminal hematuria and frequency of urination.

Examination revealed epididymitis prostatitis and left seminal vesiculitis. A urinary sinus was injected and demonstrated on the roentgenogram. This communicated with the prostatic urethra and opened on the inner surface of the left thigh.

CHONDRODYSPLASIA. DR. W. P. BLOUNT.

Chondrodysplasia was considered a rare disease in America until Ehrenfried discussed the condition in 1915 and reviewed all of the available cases in 1917. Since then many isolated examples have been reported, and Stocks has classified the forms of the disease on the basis of etiology and pathology.

A case of the disease was observed in a girl, aged 2½ years, and was followed for three years. No exostoses were present. The femora, tibiae, right fibula, left radius, pelvis, left sixth rib and the fourth middle phalanx of the left hand were locally invaded by islands of cartilage. The juxta-epiphyseal region of the diaphyses of the long bones had a cystic appearance. Bow legs and radial abduction of the left hand had been present since infancy. There was apparently no progress of the deformity. Laboratory examinations were negative. The blood calcium and phosphorus were normal. The disease appeared in no other member of the family.

CEREBRAL INJURY AT BIRTH WITH MALDEVELOPMENT AND MYXEDEMA. DR. R. L. RICHIE.

G. C., a white boy, aged 7 years, was in poor condition at birth following induction of labor by a bag. Several convulsions occurred during infancy; development was delayed. The child received thyroid substance in small amounts. On examination, the child was undersized with narrowed head in front. The skin and hair was somewhat dry and the gait somewhat spastic. Roentgen examination of the skull showed lack of space in the anterior vault. There was the low average of four carpal centers in the extremities. Examination of the blood, including the cell count, Wassermann reaction, chemical determination and Kolmer test, gave negative results. The urinalysis was negative. The dextrose tolerance test was markedly increased. The basal metabolic rate was —27 and —19 by the Benedict-Roth apparatus and —38 and —20 by the Tissot gas analysis.

The dosage of thyroid was increased gradually to 6 grains (0.04 Gm.) daily. The child has become more active physically.

INTUSSUSCEPTION WITH RUPTURE AND PERITONITIS. DR. JOHN DOCTOR.

R. A., aged 7 years, an only child, with unimportant family and past histories, on July 2 complained of pain in the stomach. The pain came in spasms, and at times, would disappear entirely for a day. The child had constipation. On July 9, it vomited but the next day felt better and played outdoors. There was no fever.

On July 12, severe pain was felt throughout the abdomen with extreme distention; the child vomited fecal matter. The temperature was 101.5 F.; the pulse rate 166, and respiration fast and grunty. The child had an anxious expression and was toxic. It lay in bed with both knees flexed and in severe pain. The eyes, nose and ears were normal. The tongue was coated. Both lungs were clear and resonant with no abnormal sounds. The heart beat was regular and bounding, with no murmurs. Examination of the urine showed: sugar ++++; no albumin; specific gravity, 1.015. The blood count was 4,100, with 40 per cent polymorphonuclears; blood sugar, 181 mg. per hundred cubic centimeters, and a negative Wassermann reaction. The abdomen showed extreme distention. No masses were felt because of this. Dr. Nott was called and operated the same evening. General peritonitis was present. About 12 inches above the cecal valve

in the ileum an intussusception was found; it was gangrenous on either side with several perforations. About 10 inches was resected, a small Murphy button inserted and anastomosis done. Three drains were inserted and closed in the usual manner. The condition of the patient was poor.

Supportive treatment was given and hypodermoclysis of physiologic solution of sodium chloride, intravenous administration of 10 per cent dextrose with insulin and morphine when necessary. The child gradually gained and improved. On July 28, hemorrhagic nephritis developed. The urine showed albumin, +++; red blood cells, ++++; leukocytes, ++. The child gradually recovered and the urine cleared up entirely. The patient was discharged on August 26, entirely recovered.

This condition probably started as an incomplete obstruction or a permeable intussusception and gradually developed further until a complete obstruction occurred with gangrene, rupture and peritonitis, Leukocytosis usually occurs early and is persistent, but this child had leukopenia. Blood and mucus was never present in the stools. After the patient was apparently on the road to complete recovery, sixteen days after operation acute hemorrhagic nephritis developed. It was thought that another obstruction had occurred, but the voiding of bloody urine explained this condition. The large amount of sugar in the urine and blood was apparently caused by irritation of the pancreas, as it cleared up four days after operation.

Program for Friday Afternoon, Sept. 27, 1929

Diabetes Insipidus; Case Report.—Dr. M. G. Peterman.
Thymic Shadows in the New-Born.—Dr. H. B. Podlasky and Dr. S. E. Kohn.
An Unusual Case of Edema.—Dr. R. M. Hall.
Teeth in Congenital Syphilis.—Dr. T. R. Abbott.
Dental Malformations and Caries.—Dr. H. H. Reese.
Medical Aspects of the Nursery School.—Dr. Bertha T. Haessler.
Anesthesia in Children.—Dr. R. M. Waters.
Congenital Hydronephrosis and Pseudohermaphrodism.—Dr. M. G. Peterman and Dr. L. B. Saffro.
Chronic Chorea.—Dr. A. I. Rosenberger.
Malaria; Case Report.—Dr. D. Koch.
Acute Rupture of the Stomach; Report of Two Cases.—Dr. Irwin Schulz.
Congenital Aural Fistula.—Dr. A. B. Schwartz.
Citric Acid Milk in Infant Feeding.—Dr. J. E. Gonce, Jr.
Rheumatic Fever.—Dr. A. E. Cohen.
Encephalitis Following Pertussis.—Dr. J. Moch.
Aphthous Stomatitis.—Dr. Arthur A. Shaeffer.
Chronic Nephritis.—Dr. R. Garens.
Malaria; Report of Three Cases.—Dr. D. Koch.
Present Status of Roentgenotherapy in Pediatrics.—Dr. H. K. Tenney, Jr., and Dr. E. A. Polile.

DIABETES INSIPIDUS; CASE REPORT. DR. M. G. PETERMAN.

A case of diabetes insipidus in a boy, aged 10 years, is presented. The father's father and the father of the patient gave a history of excessive polydypsia and polyuria with no other abnormalities. The onset was sudden with polydypsia and polyuria. Examination of the patient revealed no gross abnormalities. The urine was passed in large amounts and had a low specific gravity. The nitrogen partition showed a low total nitrogen and a low urea nitrogen. The total chlorides were also low. One cubic centimeter of solution of pituitary injected subcutaneously once in forty-eight hours gave the patient almost complete relief from polyuria and polydypsia. Solution of the posterior lobe of the pituitary body gave better results than solution of pituitary. The patient is gaining weight and developing normally.

THYMIC SHADOWS IN THE NEW-BORN. DR. H. B. PODLASKY and DR. S. E. KOHN.

This article will appear in full in the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

AN UNUSUAL CASE OF EDEMA. DR. R. M. HALL.

A normally developing, healthy girl, aged 2 years, suddenly began to vomit all food except crackers and water. In two days a transient rash appeared, followed by edema. Three specimens of urine were normal. She had gained 4 pounds (1.8 Kg.) in the previous four weeks. Pica was present.

When the child was seemingly on a high protein — low carbohydrate diet, the vomiting lessened, edema was reduced, and the weight dropped from 28 to 26¾ pounds (12.7 to 12.1 Kg.).

On a starch-free, high protein diet, there was again a reduction in edema (weight from 26¾ to 25 pounds [11.3 Kg.] and abdominal measurement, from 21 to 19¾ inches [53 to 49 cm.]). When a small amount of carbohydrate was added to the diet, the edema recurred (weight from 25 to 26¾ pounds and abdominal measurement, from 19¾ to 21 inches). In about thirty-six hours, edema of the brain occurred.

The Wassermann reaction was negative. The white blood cell count, the differential count and blood smear all were within normal limits. Numerous urinary specimens showed nothing abnormal except albumin when fever was present, and one examination showed double refractive lipoid bodies.

Blood Chemistry

	3/19/29	3/23/29
Sugar—Zolin	62.5	
Nonprotein nitrogen	44.40	33.3 mg. in 100 cc.
Urea	20.0	22.0 mg. in 100 cc.
Urea nitrogen	9.3	10.3 mg. in 100 cc.
Creatinine	2.25	
Cholesterol	....	140.0 mg. in 100 cc.

TEETH IN CONGENITAL SYPHILIS. DR. T. R. ABBOTT.

The following survey of fifty patients was made at the Milwaukee Children's Hospital:

I. *History of dental defect due to syphilis*
- A. Hutchinson, 1857
  1. Described Hutchinson incisor
- B. Moon, 1884
  1. Described mulberry molar

II. *Tooth Formation*
- A. Time teeth begin to form
  1. Lower central incisor about thirteenth to sixteenth week
  2. At birth jaws contain
     - (a) All of the deciduous teeth
     - (b) Permanent central and first molars partially formed
     - (c) Follicles for all of the permanent teeth except second and third molar

III. *Hypoplasia*
- A. May be due to any toxic or nutritional disturbance
- B. May be manifest as
  1. Cross striations
  2. Pitting
  3. Longitudinal furrowing
  4. Microdentia

C. Deciduous teeth may be hypoplastic but are not characteristic
D. Caused by syphilis may be characteristic and pathognomonic
E. Causes of in congenitally syphilitic persons
   1. General systemic disturbance
   2. Spirochete itself

IV. *Description of typical Hutchinson incisor*

V. *Description of mulberry molar*

VI. *Survey of fifty patients at the Milwaukee Children's Hospital*

DENTAL MALFORMATIONS AND CARIES. DR. H. H. REESE.

From his studies the author concludes that the problem of dental abnormalities and caries is a problem in nutrition and metabolism and not of oral hygiene. The paper emphasizes the following points:

1. The nutrition of the mother influences the structure of the teeth of her offspring and determines the incidence and extent of subsequent caries. Prevention of caries begins, therefore, with the prenatal hygiene of the mother.

2. The term delayed or difficult dentition is questioned, the author believing that all such cases can be classified as due to exudative or spasmophilic diathesis, infectious diseases, endocrine disorders, rickets, syphilis and feeblemindedness.

3. The various discolorations of teeth are not to be regarded as the result of poor oral hygiene entirely. Greenish discoloration about the neck of the upper incisors is seen in children suffering from nutritional disturbances, to be followed later by caries; brown spots on the surfaces of incisors and cuspids are not iron deposits from drinking water but are the residuals of damage to the structural anlage.

4. The teeth seen in ricketts are delayed, irregular and asymmetrical. Upper incisors are dwarfed, and horizontal grooves or pits are frequent. Caries appears at the margins early. These teeth are usually yellowish. The alveolar processes also show the lack of calcium and vitamin D, and malocclusion results from the muscle pull on soft bony structures.

5. The author calls attention to the predominance of dental disorders in the young and especially the occurrence in the periods of puberty and pregnancy, thus indicating a relation of the thymus and parathyroids, and the debilitating influences of syphilis on the endocrine system are stressed.

MEDICAL ASPECTS OF THE NURSERY SCHOOL. DR. BERTHA T. HAESSLER.

The aspects of the nursery school presented are: development of the school in England and in America: its relation to the day nursery and the kindergarten; aim of the nursery school; nursery school procedure; the physical environment; play equipment; food; training in eating habits; naps; attendance — influenced by infectious disease; intelligence tests; advantages of nursery school to the child; parental education; the nursery school as an aid to the physician.

ANESTHESIA IN CHILDREN. DR. R. M. WATERS.

Anesthesia in children presents certain difficulties not commonly met in adults. The induction of anesthesia in the young is often difficult of accomplishment without considerable crying and struggling.

Tribromethylalcohol (CBr_3CH_2OH) has been studied clinically as a possible anesthetic agent in Germany during the past four years. Investigations, both laboratory and clinical, at the Wisconsin General Hospital and at the Medical School of the University of Wisconsin have extended over the past fifteen months.

The induction of anesthesia by the rectal instillation of this drug accomplishes a surgical anesthesia with surprisingly little opposition from the patient, and for this reason it was presented to the Central States Pediatric Society for con-

sideration. It is subject to the serious criticism that the dose must be predetermined, and after once having been administered, is out of control. Small doses short of full anesthetic doses and added supplemental anesthesia with a rapidly controlled agent such as nitrous oxide are recommended. A complete account of the work with this drug at the University of Wisconsin, with the technic used, and a statistical analysis of the results will appear later.

CONGENITAL HYDRONEPHROSIS AND PSEUDOHERMAPHRODISM. DR. M. G. PETERMAN and DR. L. B. SAFFRO.

A white infant, aged 1 day, on examination revealed pseudohermaphrodism with prominent abdomen and clubfoot. Roentgen examination showed gas-containing bowel in the upper and left portion of the abdomen, leaving the right and lower part with solid shadow. Abdominal paracentesis was done a number of times and clear straw-colored fluid removed each time. About five weeks after admission

Fig. 1.—Hydronephrotic kidney from child with pseudohermaphrodism.

a definite smooth tumor mass could be felt in the lower right quadrant. Operation was not deemed advisable because of the child's poor general condition. Death occurred eighty-eight days after admission. Postmortem examination showed the presence of a large hydronephrotic kidney in the right lower quadrant, complete obstruction of the right ureter, and ovaries and tubes but not uterus.

CHRONIC CHOREA. DR. A. I. ROSENBERGER.

The chief object in presenting this case was to show the possibility of chronic chorea developing in a child, aged 7 years.

Until recently, little was known concerning the pathology of the choreas. Many of modern textbooks of pediatrics fail to mention lesions of the striate bodies as the possible cause of choreic movements.

This child, who had generalized choreic movements, was mentally defective, with the age of certainly not more than 4 years.

The first thought that naturally occurs to the pediatrician as the cause of this condition is injuries occurring at birth. Dr. Rosenberger, however, takes the position that trauma cannot possibly be so selective as to involve only the striate bodies.

Huntington's chorea and Wilson's disease can be ruled out, as they are both diseases of adult life. In the one, there is a hereditary background and in the other, cirrhosis of the liver.

Congenital syphilis is another possibility. Dr. Camp of Ann Arbor, Michigan, has reported a case of chronic chorea the result of congenital syphilis in which the pathologic lesion was undoubtedly in the striate bodies. In this case, however, there is no evidence of congenital syphilis.

It is possible that the condition was the result of an encephalitis residue. A note made by the attending pediatrician shortly after the birth of the child, mention increasing drowsiness. There was also fever on the third day, marked generalized jaundice, a high white cell count, albumin 3 plus, and blood in the stool.

Every one is familiar with disorders of the extrapyramidal tract in chronic encephalitis or following encephalitis. It is also known that encephalitis is extremely common in early childhood.

A picture of chronic chorea in a child, aged 7 years, first noted at 6 months of age, is presented. The disorder is not typical of any disease of childhood. It is probably the result of encephalitis with either degenerative or inflammatory lesions in the striate bodies of the brain. The prognosis is bad, and no treatment is of any avail.

ACUTE RUPTURE OF THE STOMACH: REPORT OF TWO CASES. DR. IRWIN SCHULZ.

CASE 1.—S. L. F., aged 17 months, entered the hospital on Feb. 5, 1929, complaining of vomiting and pain in the abdomen of four days' duration. On examination, the temperature was 102 F. The child was acutely ill. The abdomen was distended, with visible peristalsis associated with pain. Anemia and leukocytosis were present. Death occurred thirty-six hours after entry.

Autopsy revealed terminal pneumonia; intestinal obstruction, and rupture of the stomach with early peritonitis.

CASE 2.—W. S. M., aged 3 years, entered the hospital on Aug. 3, 1929, complaining of fever and labored breathing of twenty-four hours' duration. The child had had convulsions just previous to entry.

On examination, the child appeared toxic and breathing was labored. The tonsils were hypertrophic and an angry red, with patches of white exudate and bleeding on manipulation. The neck was stiff. The temperature was 103; pulse rate, 112, and respirations, 40. The child developed signs of meningitis, and on the third days, respiratory paralysis. Artificial respiration was instituted, but, death occurred soon after.

Autopsy revealed: encephalitis; acute splenic tumor; hydro-ureter (left), ruptured stomach at the junction of the esophagus, and beginning peritonitis in upper left quadrant.

CONGENITAL AURAL FISTULA. DR. A. B. SCHWARTZ.

At one of the 1928 meetings of the Chicago Pediatric Society, Parmelee presented two instances of fistula auris congenita in the new-born. Two similar cases, one showing a unilateral, the other a bilateral form of this anomaly in older children are presented.

Congenital aural fistula is recognized as a small, almost unnoticed opening of the skin usually situated about 1 cm. above the tragus and about 2 or 3 cm. in front of the anterior end of the helix. It is not a true fistula but a culdesac running obliquely forward, from 0.5 to 1.5 cm. deep. Usually, the canal exudes a slight milky secretion and is open to the possibility of infection and formation of fistulas.

According to Seifert (Ueber die Sogenannte Fistula auris congenita, *Deutsche Ztschr. f. Chir.* **209**:118, 1928) Virchow's theory, relating the anomaly to an imperfect closure of the first branchial cleft, is not tenable. The case on which

Virchow predicated his argument was truly such an example. However, it was one involving other structural anomalies, such as absence of the external ear.

Seifert suggested the term fistula auriculae congenita. He regarded it as an imperfect closure of the ear muscle alone, occurring at some time between the fourth and fifth embryonal month.

A similar embryologic basis explains those instances in which instead of a pit, a projecting tubercle is noticed.

The anomaly has been recorded as a familial characteristic, and Ruttin (Zur Frage der Fistula auris congenita und der Aurikularanhänge, *Wien. med. Wchnschr.* **77**:1019, 1927) described a family in which certain members presented the fistula type, others, the tubercle type of anomaly.

Citric Acid Milk in Infant Feeding. Dr. J. E. Gonce, Jr.

This article will be published in full in a later issue of the American Journal of Diseases of Children.

Rheumatic Fever. Dr. A. E. Cohen.

A girl, aged 8 years, had an unexplained fever and an intermittent rash of four months' duration when finally symptoms more clearly associated with acute rheumatic fever made their appearance and cleared up the diagnosis. The patient made an uneventful recovery after antirheumatic treatment was instituted and the tonsils were removed.

Encephalitis Following Pertussis. Dr. J. Moch.

J. W., a white boy, aged 12 months, was admitted to the isolation ward on Aug. 9, 1929. A diagnosis of pertussis of five weeks' duration complicated with encephalitis and bronchopneumonia was made. The latter condition rapidly improved with treatment, but the lethargic stage persisted for five weeks without signs of improvement. During that period the infant recognized neither objects nor people and was hyperirritable. Feedings had to be forced for a short period. Fairly rapid return to a nearly normal mental condition occurred during the last two weeks in the hospital.

Aphthous Stomatitis. Dr. Arthur A. Schaefer.

A case is reported of aphthous stomatitis complicated by skin lesions due to hemolytic streptococci which were isolated from the milk supply.

Chronic Nephritis. Dr. R. Garens.

A boy, aged 12 years, was admitted to the Milwaukee Children's Hospital with complaints of weakness and recurrent attacks of hematuria with no history of edema, increase in blood pressure or abnormalities of the fundi. On physical examination the boy was pale and thin; the tonsils were diseased; the teeth decayed, and the spleen and right kidney palpable.

During his stay in the hospital, he developed edema, formed elements in the urine; casts and doubly refractive lipoid bodies, and pus and red cells. The blood count revealed secondary anemia. There was no improvement in the child's condition, and death occurred.

Postmortem examination revealed generalized edema; free fluid in the pleural and abdominal cavities; a thick, adherent pericardium, and fetal lobulations of both kidneys, the cortex of which were thin and light, with hemorrhagic spots throughout, and the medulla of which appeared pale and yellow.

Malaria; Report of Three Cases. Dr. D. Koch.

There were three cases of malaria in the Milwaukee Children's Hospital in August: one in a boy, aged 11 years, with meningeal symptoms; one in a girl, aged 3 years, with symptoms of an acute condition of the abdomen; one in a

boy, aged 10, with a typical malarial history. Tertian malarial parasites were demonstrated in blood smears of each. The temperature curves were all typical, and quinine medication allayed the symptoms and chills successfully. Only one case was acquired in Milwaukee, the other two patients being from Red Wing, Minnesota, and Madison, Wisconsin.

PRESENT STATUS OF ROENTGENOTHERAPY IN PEDIATRICS. DR. H. K. TENNEY, JR. and DR. E. A. POHLE.

The following biophysical principles of irradiation are pointed out:

1. Young, active cells are more susceptible than mature ones.

2. The effect of irradiation is destructive no stimulating effect having been demonstrated.

3. The skin of children is no more susceptible than that of adults, but the relation of skin area to body volume is important.

The following are discussed:

1. The glands of internal secretion. The rôle of the thymus is still uncertain but may be of considerable importance in producing many annoying symptoms.

2. Tuberculosis, especially of the glands.

3. Blood diseases.

4. Skin diseases.

5. Inflammatory conditions, especially acute cervical adenitis.

Program for Saturday, Sept. 28, 1929

Osteomyelitis.—Dr. F. J. Gaenslen.
Atresia of the Esophagus.—Dr. S. M. B. Smith.
Experimental Acute Glomerulonephritis in Rabbits.—Dr. L. M. Warfield.
Pertussis Vaccine in Prophylaxis.—Dr. R. F. Schowalter.
Resuscitation of the New-Born.—Dr. R. S. Cron.
Tuberculosis in Children.—Dr. K. E. Kassowitz.
Tonsils and Nutrition.—Dr. Eleanore Cushing-Lippitt.
Bone Changes in Continuous Phosphorus Ingestion.—Dr. Helen J. Zillmer.
Rheumatic Fever.—Dr. N. Enzer.
Congenital Syphilis.—Dr. S. M. Markson.
Institutional Infections and Prophylactic "Cold" Vaccines.—Dr. F. R. Janney.
Acute Mediastinal Abscess.—Dr. B. J. Malnekoff.
Tuberculosis in Children from the Sociologic Point of View.—Dr. A. A. Pleyte.
Spontaneous Gangrene of Both Legs.—Dr. L. D. Smith.

OSTEOMYELITIS. DR. F. J. GAENSLEN.

This subject deserves discussion because lack of early diagnosis is followed by disastrous results. The fact that skin infections, often apparently trivial, such as infected blisters or hangnails, are known to be important factors in the causation of osteomyelitis puts responsibility on the physician. By directing the attention of the public to the need of careful attention to even minor infections, a good deal may be done in a prophylactic way to cut down the incidence of this disease.

It is rare that the orthopedic surgeon sees cases of osteomyelitis within the first twenty-four or forty-eight hours. If proper attention is given in this early period, many of the calamitous results could be avoided. Of the three types, acute hematogenous osteomyelitis, that due to extension from neighboring infection, and that due to direct infection as in gun shot wounds, only the first group will be here considered.

Organisms reaching the general circulation are carried through the nutrient vessel to the cancellous bone near the epiphyseal line. Slight trauma by breaking

down a few trabeculae and causing slight hemorrhage often determines the localization of the infectious process. Once established, the infection spreads through the cancellous bone and through the cortex which is very thin near the epiphysis to beneath the periosteum, causing intense continuous throbbing pain. The diagnosis as a rule is readily made. The exceptional case offers much difficulty.

Careful examination should include inspection, palpation, motion of the neighboring joints, mensuration, laboratory, and roentgen observation. Inspection in the early stages will reveal no local changes. The information obtained by careful palpation is most important. There is tenderness near but not over the joint. Motion within a limited range is without pain. These two points are of great value in differential diagnosis of osteomyelitis from acute articular rheumatism. In the first day or two there is no atrophy and, therefore, mensuration gives little information. The leukocyte count is high as is the percentage of polymorphonuclears in the differential count. I would almost advise against roentgen examination in the early stages for fear that negative manifestations might be construed as evidence against osteomyelitis. If one is in doubt, an incision can do but little harm in a case of rheumatism compared with the great advantage of early drainage in osteomyelitis. Once the diagnosis is made, drainage should be immediate. An overnight delay can find no justification. It is not improbable that under certain conditions an infection of the blood stream as massive as if a culture were injected intravenously with a hypodermic syringe may occur. Treatment should consist in incision to relieve tension.

The Orr method of petrolatum pack and the plaster cast in the after treatment are advocated. The petrolatum pack is not a plug, but acts as a huge drain, the discharge seeping out between the petrolatum and the surface of the wound. It also supplies that optimum of pressure conducive to rapid healing. The plaster cast prevents meddling, reinfection, and the breaking down of nature's barriers in defense.

Aside from this the freedom from pain and the absence of the dread of repeated dressings must make the method popular, leaving out of consideration entirely the convenience of the surgeon who will find the method time saving.

From the standpoint of prophylaxis, it should be mentioned that the general practitioner can do much toward reducing the incidence of this disease by educating his clientele to pay strict attention to so-called minor skin infections, as it is well known that infected blisters, hang-nails, and furuncles of the skin are a frequent cause of osteomyelitis.

ATRESIA OF THE ESOPHAGUS. DR. S. M. B. SMITH.

Congenital atresia of the esophagus is of infrequent occurrence and is usually connected with other congenital malformation, probably the trachea and the bronchi being the most common, into which the lower pouch opens. Congenital stenosis and congenital dilatation are occasionally found as well as the separation of the esophagus into upper and lower noncommunicating blind pouches. Persistent and nonprojectile vomiting in the new-born, after a few cubic centimeters of water has been given and there has been no evidence of any peristaltic waves in the abdomen, may arouse suspicion.

The case reported was that of the second child, a girl, of white parents both of whom were of average health. There was no history of anything other than the ordinary diseases of childhood. The mother had had two miscarriages, one before and the other following the birth of this child, and both of accidental origin. The Wassermann reaction of her blood was negative. The prenatal period before the birth of this child was uneventful and the confinement fairly easy. The infant was apparently normal, weighing 7 pounds and 2 ounces (3,231.8 Gm.) and cried lustily immediately following delivery. The heart and lungs were normal. The bladder and bowels emptied within a few minutes. About six hours later, two teaspoonfuls of warm water was given; this was regurgitated within a few minutes, and in about an hour more water was given with similar results. After

several repetitions of this, an attempt to pass a catheter into the stomach was made without success. A small dose of barium was given, and the roentgenogram showed a pouch about 2½ inches (6.2 cm.) in length and no evidence of any barium passing below that point. The infant died on the eighth day, and post-mortem examination confirmed the diagnosis. There was an entire absence of esophageal tissue between the two pouches, but they were connected by several small fibrous bands. In this case there was no connection with either the trachea or the bronchi.

There was another interesting case a few weeks later in the hospital of complete atresia of the lower portion of the descending colon; the infant, a boy, died about one week after birth, and autopsy revealed the true condition. Owing to an accident at the time of mounting, the specimen was rendered unfit for use. The author has been unable to find any reports of this malformation in the literature.

EXPERIMENTAL ACUTE GLOMERULONEPHRITIS IN RABBITS. DR. L. M. WARFIELD.

The work reported is an attempt to repeat the work of Duval and Hibbard who in 1926 published their results on the production of acute glomerulonephritis in rabbits. The method they used was to immunize rabbits against streptococci of scarlet fever, then to inject into the peritoneal cavity of the immunized rabbits large masses of streptococci, to remove the resulting exudate, filter through a Berkefeld filter and make intravenous injections into nonimmune rabbits.

The pathologic anatomy of the kidneys of rabbits treated by this procedure is also reported.

PERTUSSIS VACCINE IN PROPHYLAXIS. DR. R. P. SCHOWALTER.

It is important to know whether or not prophylactic injections of whooping cough vaccine prevent the disease.

Convalescent serum was used to control and prevent whooping cough as early as 1901. Vaccines have been used for more than sixteen years, and a review of the literature reveals many conflicting reports. A study of the statistics from the Milwaukee County Home for Dependent Children is presented. In the past eight years, all children under 6 years of age were given prophylactic injections on entrance to the institution. More than 2,700 children received such injections. The incidence of whooping cough during this period is compared with the incidence in the eight preceding years during which no vaccine was used.

In the eight year period during which no vaccine was used, 161 cases of whooping cough developed at the institution, the morbidity rate being 67.5 per 1,000 children. During this "no vaccine" period, 8 deaths were attributed to whooping cough.

During the "vaccine" period, 1921 to 1928, inclusive, there were 377 cases of whooping cough, the morbidity rate being 136.4 per 1,000 children. During this period, there were 11 deaths due to whooping cough.

Pertussis vaccine was unable to prevent an increase in the number of cases of whooping cough. Such an increase was to be expected, however, as Milwaukee, where most of the children in the institution come from, experienced a similar increase during this eight year period.

Both the City of Milwaukee and the County Home for Dependent Children had a lower mortality rate for whooping cough from 1921 to 1928 than in the preceding eight years.

RESUSCITATION OF THE NEW-BORN. DR. R. S. CRON.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

TUBERCULOSIS IN CHILDREN. DR. K. E. KASSOWITZ.

The first stage of the bronchogenic tuberculous infection consists of the primary focus and the corresponding lymphadenitis. In certain cases infiltrations of the intermediate parenchyma between focus and lymph nodes become visible on the roentgenogram but scarcely audible on physical examination. These early lesions with the characteristics of exudative infiltrations have been interpreted as allergic tissue reactions probably due to exogenous reinfection. They undergo at times retrogressive changes and may even disappear completely without leaving any visible scar on the roentgenogram. In other cases, however, the early allergic infiltrations may progress to a true phthisic process, which fact has lead to the new hypothesis of a closer connection between the juvenile and the adult type of pulmonary tuberculosis.

TONSILS AND NUTRITION. DR. ELEANORE CUSHING-LIPPITT.

A review of 12,000 records of physical examinations of working children attending vocational school part time was made. In making the calculations that

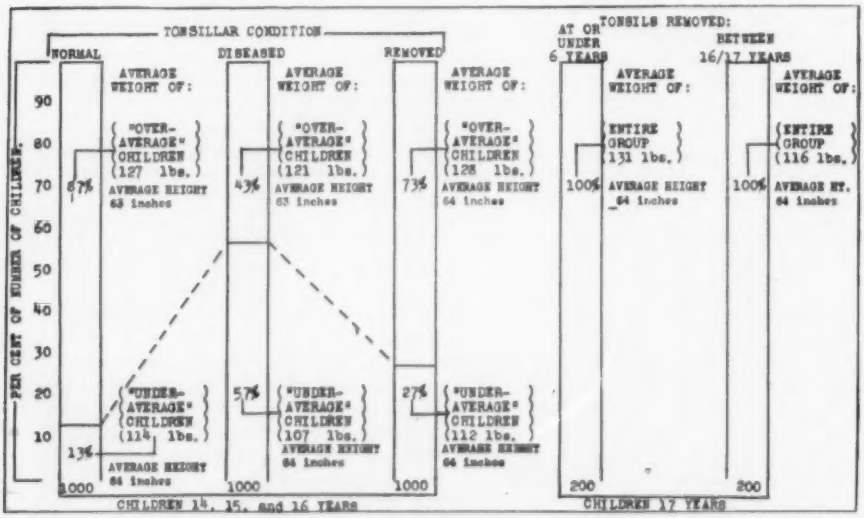


Fig. 2.—Record of study of tonsils and nutrition of 3,400 working children of Milwaukee in part time attendance at school. Average as used, is based on standard age, height and weight chart.

sum up the observations relative to the nutrition of the children, all cases were excluded in which pulmonary conditions, hyperthyroidism and other diseases affecting nutrition were or had been present or suspected to be present. Cases showing dental defects could not be excluded, as few children have continuous and adequate dental care.

BONE CHANGES IN CONTINUOUS PHOSPHORUS INGESTION. DR. HELEN J. ZILLMER.

A series of observations of the hands, wrists, knees, ankles, and other bones extending over a period of two years, and recorded by means of roentgenograms was reported in a boy with tuberculosis of the bone who had been taking phosphorus in cod liver oil consistently for nineteen months prior to the time of this series. The dense phosphorus zone was first seen as a compact wide band parallel to

and at the epiphyseal line, broadest about the knees, wrists and ankles, where growth is rapid, and narrower but distinct in other bones where growth occurs slowly. The phosphorus band was first described by Wegner in 1872. Because of the marked atrophy of all the bones in the case presented, the phosphorus band was especially strikingly seen. As with other transverse bands, its fate is to move gradually shaftward away from the metaphysis, as here recorded, until it is seen as a fading band well up in the bone shafts at the end of two years.

RHEUMATIC FEVER. DR. N. ENZER.

Illustrations are presented of the acute changes in rheumatic fever as shown by the diffuse infiltration of the myocardium, aorta, pulmonary artery and pericardium; demonstration of acute changes in the blood vessels of the heart; illustration of older and chronic lesions, Aschoff bodies, and scar formation. Brief mention is made of experimental efforts to produce this disease in animals.

CONGENITAL SYPHILIS. DR. S. M. MARKSON.

Only a small percentage of heredosyphilitic infants manifest the frank multiform symptoms described in textbooks; usually symptoms are solitary and tend to imitate other diseases. In a large percentage of cases, the condition tends to remain latent. The present day thought holds that Colle's law is invalid and that all heredosyphilitic patients are infected in the womb by spirochetes carried there through the maternal blood stream.

Knowledge of etiology is valuable from the prophylactic standpoint. Ten per cent of pregnant women show positive Wassermann reactions. Wassermann reactions of the blood taken in infants at birth are unreliable. They show too many negative results which in twelve or fourteen days may become positive. On the basis of symptoms alone, without check-up of a Wassermann reaction on the infant and family, diagnosis in only 50 per cent of the cases is likely. Treatment of syphilitic pregnant women during each pregnancy will reduce likelihood of infection in infants to approximately 8 per cent.

INSTITUTIONAL INFECTIONS AND PROPHYLACTIC "COLD" VACCINES. DR. F. R. JANNEY.

The control of upper respiratory infections and the contagious diseases in nonmedical institutions for children is inadequate. A lack of understanding of control of contagion by the lay staff, frequent visitors and the ward system with a large number of children in close contact all contribute.

Respiratory infections and their complications are much more prevalent among the younger institutional children than among the older ones. The children under 3 years of age in a 120 bed denominational home averaged some evidence of a respiratory infection for approximately one-half the time of an observation period of eight months. The older children had somewhat more respiratory infection than children in their own homes would be expected to have.

Six weekly doses of a standard stock cold vaccine totaling 2.1 cc. were given in the fall to thirty of the children in institution mentioned. The six of these under three years of age averaged more respiratory infections for the eight month period of observation (130 days to 105 days for the control group) than did their control group. The twenty-four older children given the vaccine averaged thirteen days of respiratory infections for the observation period, while their control group average twenty-four days. The older girls had fewer days of illness than did the older boys.

ACUTE MEDIASTINAL ABSCESS. DR. B. J. MALNEKOFF.

This article will be published in full in a later issue of the AMERICAN JOURNAL OF DISEASES OF CHILDREN.

TUBERCULOSIS IN CHILDREN FROM THE SOCIOLOGIC POINT OF VIEW. DR. A. A. PLEYTE.

There has been a 50 per cent reduction in the death rate from tuberculosis in the last twenty years. From 60 to 95 per cent of children in Vienna between the ages of 11 and 14 years were infected, according to earlier studies. At that time, tuberculosis caused 20 per cent of all deaths. Recent studies showed from 10 to 65 per cent of children under 18 years of age infected.

Today, tuberculosis causes only about 7 per cent of all deaths in this country.

The lower percentages of infection occur in rural communities. With a lower death rate has come also a lower incidence of infection. The childhood and occult types of tuberculosis furnish many of the future cases of open tuberculosis. Children usually acquire the disease from tuberculous parents. The childhood type of the disease comprises that group of individuals, usually children, who show a preponderance of tuberculosis in the tracheobronchial glands, although there may be, or there may have been, slight involvement in the lungs or other parts of the body. A diagnosis of presumptive disease is made by a carefully taken history, thorough physical examination, temperature and pulse study, tuberculin reactions and stereoscopic roentgenograms of the chest.

One can rule out tuberculosis in diagnosis in more than 50 per cent of the children seen today under 15 years of age by so simple a procedure as a tuberculin test.

Pediatricians must have frequent use for this test. Few tests are more simple, less time consuming, less expensive and, on the other hand, of more value in diagnosis. One should always be suspicious of tuberculosis, knowing it is still so frequent an infection and so common a disease. Tuberculosis will cease to be a plague to mankind only when pediatricians, general practitioners and tuberculosis and health workers coordinate their efforts. Five means of approach on which they can cooperate, from a clinical and sociologic point of view are:

1. Isolation of Adults with Tuberculosis.—Grandparents, parents, brothers, sisters, servants or other persons in the home may have tuberculosis and be a source of infection to the child. If tuberculosis is suspected in a child, adult members of the home should be examined and, if found to be tuberculous, isolated. Tuberculosis most frequently starts in childhood, and the child is usually infected by its nearest relatives.

2. Educational Campaign.—Adults must be taught the common early symptoms of tuberculosis. They must be persuaded to cover cough and sneeze, no matter what the cause may be. Sometimes the cough is due to the tubercle bacillus. The public as a whole and individually should receive continual guidance on how to live rightly. Children need intensive teaching, training and practice in the performance of health chores to the point where health habits are established. Periodic examination of adults and children by the attending physician must become as common and natural an event in the lives of the people as is dental examination.

3. Early Suspicion of Tuberculosis.—No age group is free from tuberculosis. Few families are untainted. Twice as many active cases of phthisis exist unknown to themselves or anyone else as are known to all health agencies combined. Tuberculosis manifests itself in so many different ways that its onset may simulate other diseases closely. Hence, one must always suspect its presence in differential diagnosis.

4. Careful Study of Children.—Such a study includes a painstaking history, a thoughtful examination, an accurately taken record of temperature and pulse, tuberculin tests and particular stereoscopic roentgenograms of the chest. Only by paying attention to details as children are watched may one's suspicions be aroused.

5. Treatment Until Safe.—Because safety is a relative term as applied to this disease and will be determined largely by the patient's mode of life, one can never be sure that a patient is cured. One must, then, be conscious of the possibility

of tuberculosis in all families. One must watch, too, all infected children, not only through their childhood but through adolescence, and even adult life.

Tuberculosis should be suspected early. Children must be studied carefully, and all diagnostic methods available should be used as far as practicable. Observation and treatment must be prolonged. Seldom has treatment been continued long enough — frequently patients have been discharged or have discontinued treatment too soon. These patients should be followed as long as possible. By observing these suggestions one can best learn to know tuberculosis from its inception until it is arrested.

SPONTANEOUS GANGRENE OF BOTH LEGS. DR. L. D. SMITH.

A girl, aged 6 years, of Italian parentage, was in normal health until the age of 3 years, when she fell down stairs; after that she was subject to convulsions. She was born at full term; labor was difficult; she weighed 3 pounds (1,360.78 Gm.) paralysis of the left arm cleared up spontaneously.

In January, 1929, while under observation for the epileptiform seizures, she contracted a respiratory infection. The parents insisted on removing her from the hospital against advice. She walked down stairs, was taken home in a taxicab, when the temperature was 2 below zero. That night she screamed with pain in both legs which became reddened and somewhat swollen, with brownish spots extending about two thirds of the distance up the leg. In a few days both limbs became dry and black. A line of demarkation established itself in both legs at about the junction of the middle and upper thirds of the tibia. Bilateral amputation was performed at one sitting in March. The stumps promptly healed, and the child made an uneventful recovery and is well now with the exception of the epileptiform seizures which still continue.

Dissection of the veins and arteries on the amputated limbs was difficult because of sticky adherent tissue surrounding the vessels. There were no marked changes within the vessel walls. The arteries and veins both contained occluding thrombi.

There seem to be several factors in operation here each of which in numerous other cases has been associated with gangrene. Blood changes with formation of thrombi in association with various infections, especially lung infections, is not uncommon. Epilepsy, epileptiform seizures and convulsions may have as a contributing element a local vascular spasm that in some of its manifestations is called Raynaud's disease. The relationship of exposure to low atmospheric temperatures to neurovascular changes is well recognized.

These factors may have contributed, and probably did contribute, to the development of this condition; however, in spite of the age and sex of the patient, in view of the state of the tissue, it would seem that this is a case of thrombo-angiitis obliterans bilateral, with a definite acute onset.

Book Reviews

APPLIED ELECTROCARDIOGRAPHY. By A. E. PARSONNET and A. S. HYMAN. Price, $4. Pp. 192, with 114 illustrations. New York: The Macmillan Company, 1929.

Electrocardiography is not merely a contribution to laboratory physiology; it is a valuable practical method of the diagnosis of heart disease. It ranks well in importance with the roentgen ray and the sphygmomanometer. The practitioner is inclined to look on the electrocardiograph as a mysterious machine which makes mysterious curves of only academic importance. As a matter of fact, the clinician can obtain much practical help from it in the diagnosis of circulatory diseases and rheumatism. He will, therefore, do well to familiarize himself with the indications for an electrocardiographic study, and to be able to correlate the information presented in the graphic records with his clinical observations.

The criticism which can be made against most medical books cannot be made against this monograph, namely, too long a distance between the covers. The authors have written clear of all debatable rubbish. They give the reader a clear idea of the physiology of the subject, a description of the apparatus, its value and its limitations. The book is compact, well ordered and simple in style and shows excellent sequence. I wish to emphasize only one of the many important and practical points which the authors bring out in their monograph. If after an infectious disease the convalescence is unusually delayed, an electrocardiogram should be taken; for this delay is often due to disturbances of conduction caused by toxic damage of the heart muscle. Serious and often fatal complications to the heart may thus possibly be prevented.

THE BLOOD PICTURE. By PROF. DR. VICTOR SCHILLING. Translated and Edited by R. B. H. GRADWOHL, M.D. Seventh and eighth revised edition. Price, $10. Pp. 408, with 4 colored plates and 44 illustrations. St. Louis: C. V. Mosby Company, 1929.

This book is not intended to replace the well known manuals of hematology, but to supplement them in a practical way, by simplifying existing methods. The first part deals with the technic of blood examination, and only those methods are given that are easily applicable. The second part is given to the theory, morphology and division of the blood picture. It is rather unfortunate that in a work of this kind more attention could not be paid to the English and American literature. This is particularly noticeable in the discussion of the work of Arneth in which nothing is said of Cooke's polymorphonuclear classification, or in the chapter on the blood in the new-born infant in which no mention is made of the studies of Lucas. The third part of the book deals with the principles for the clinical use of the blood picture, and the fourth selected examples for practical use. The last two parts are intensely interesting. The clinical cases are discussed in full, and the resulting blood pictures are outlined. These are illustrated by the author's method of "hemograms." In this system a simple classification of the polymorphonuclears is used, the division being into myelocytes, juveniles, stabs and segments. This is infinitely more practical than the cumbersome classification of Arneth. This book is well written and readable. A full bibliography of the German literature has been added. The printing and binding are excellent, and the paper is of exceptionally good quality. This book should be of value to every internist who is interested in hematology.

DISEASES OF WOMEN: SYMPTOMS AND TREATMENT. By FRANKLIN I. SHROYER. Price, $2. Pp. 103. Boston: The Gorham Press, 1929.

The purpose of this rather brief book of about 100 pages is not entirely clear to the reader. It contains information not suitable for the lay reader. It is too

sketchy for the student and it contains little that the practitioner does not know. It is not well written and contains many errors both in spelling and in grammatical construction. It contains chapters on a variety of subjects such as the gynecologic examination which seems to be written more for the lay reader. The next chapter on the diagnosis and treatment of tumors is rather technical and beyond the lay reader, but is not of great interest to those who are medically educated. The malignant conditions of the uterus are also considered, and their treatment with radium is considered in a separate chapter, which is about the best in the book.

Other topics considered in separate chapters are the morbid conditions of pelvic organs in women and their relations to various nervous disorders; diet with mention of ideally balanced menus; chronic metritis and allied conditions; infections of the uterus and cervix and their treatment, and a discussion of tumors of the breast.

The book seems to carry a possible appeal to both the lay and the professional reader with the probability that it will not prove satisfactory to either.

THE NUTRITION OF HEALTHY AND SICK INFANTS AND CHILDREN. By NOBEL, E., C. P. PIRQUET and R. WAGNER. Translated by BENJAMIN M. GASUL. Price, $3.50. Pp. 243, with 78 illustrations (including charts). Philadelphia: F. A. Davis Company, 1929.

The first part of the book gives an excellent exposition of Pirquet's system of nutrition. At first, this strikes me as unnecessarily complex, but when once mastered along with the key letters for description, one can see its advantage — particularly in dealing with large numbers of infants and children. Calculations for diet are based on the supposed fact that the square of the sitting height represents the absorbing area of the intestine, and that the maximum amount of food that can be handled in twenty-four hours is the equivalent of 1 cc. of breast milk per square centimeter of intestinal surface. The minimum is three tenths of this amount; the optimum usually seven tenths.

The larger part of the book discusses the actual management of individual cases. Complete graphic charts accompany each history. The nutritional management of such special conditions as Herter's infantilism, cyclic vomiting, kidney diseases, diabetes, epilepsy and anemias are discussed separately. The last chapter contains diet forms and recipes.

MODERN METHODS OF FEEDING IN INFANCY AND CHILDHOOD. By D. PATERSON and J. F. SMITH. Price, $3. Pp. 106. New York: Paul B. Hoeber, Inc., 1929.

The first part of the book has to do with foods used in infant feeding. Considerable space is given to all types of patent foods, and while this is excellent for reference, one feels that their importance is overemphasized. The discussion of practical considerations in breast feeding is excellent. In the chapter dealing with the modification of cows' milk, the physiology of digestion is touched on in relation to the different elements of cows' milk. Suggested menus for the feeding of older children are given.

The last part of the book discusses vomiting, diarrhea and constipation, the atrophic and the premature infant and diets to be used in different diseases of infancy and childhood. Although this book is full of practical suggestions, rather too little attention is paid to physiology and physiologic pathology to make it of great value to the medical student.

PHYSICAL MEASURES OF GROWTH AND NUTRITION. By RAYMOND FRANZEN, PH.D. Paper. Price, $1. Pp. 138, with 1 illustration. New York: American Child Health Association, 1929.

This is the second monograph in the School Health Study Series. The source of material is from measurements of 7,500 school children. The monograph is

devoted to this form of measurements, and an analysis of the anthropometrical data of the study. The measures may be of value as research devices, but their practical use is doubtful.

THREE MINUTE MEDICINE. By LOUIS R. EFFLER. Price, $3. Pp. 453. Boston: The Gorham Press, 1929.

This book contains a collection of newspaper sketches.

Directory of Pediatric Societies

INTERNATIONAL

International Congress on Pediatrics

Prof. I. Jundell, Artillerigatan 23, Stockholm, Sweden.
Place: Stockholm. Time: Aug. 17-20, 1930.

NATIONAL

American Medical Association, Scientific Assembly, Section on Diseases of Children

Chairman: Dr. William Weston, 1231 Bull. St., Columbia, S. C.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Detroit. Time: June 23-27, 1930.

American Pediatric Society

President: Dr. Joseph Brennemann, 30 N. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: Windsor Hotel, Montreal. Time: June 2-4, 1930.

The Association of American Teachers of the Diseases of Children

President: Dr. Lawrence T. Royster, State Board of Health, University, Va.
Secretary: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.

American Child Health Association

President: Hon. Herbert Hoover, 370 7th Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 7th Ave., New York.

Canadian Society for the Study of Diseases of Children

President: Dr. Thomas Crossan Clark, 51 Herkimer St., Hamilton, Ont.
Secretary-Treasurer: Dr. Frank H. Boone, 164 James St. S., Hamilton, Ont.

SECTIONAL

Central States Pediatric Society

President: Dr. Edward Clay Mitchell, 1073 Madison Ave., Memphis, Tenn.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Ave., Chicago.
Place: Memphis, Tenn. Time: Fall, 1930.

Section on Pediatrics of Southern Medical Association

Chairman: Dr. H. Leslie Moore, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. Ludo von Meysenbug, 3501 Prytania St., New Orleans, La.
Place: Louisville, Ky.: November, 1930.

LOCAL

Alabama Pediatric Society

President: Dr. J. MacBell, Mobile.
Secretary-Treasurer: Dr. Hughes Kennedy, Jr., Highland Plaza Apts., Birmingham.

Northern California Pediatric Society

President: Dr. Edward B. Shaw, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Lloyd Hardgrave, 384 Post St., San Francisco.
Place: Various teaching schools. Time: Monthly.

Southwestern Pediatric Society

President: Dr. Ezra S. Fish, Pacific Mutual Bldg., Los Angeles.
Secretary: Dr. Fred L. Glascock, 1136 W. 6th St., Los Angeles.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

The Rocky Mountain Pediatric Society

President: Dr. Elsie Seelye Pratt, Republic Bldg., Denver.
Secretary-Treasurer: Dr. Wilford W. Barber, Metropolitan Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month from September to May, inclusive, 2:30 p. m.

Georgia Pediatric Society

President: Dr. W. N. Adkins, Medical Arts Bldg., Atlanta.
Secretary-Treasurer: Dr. E. N. Gleaton, 212 E. Gaston St., Savannah.

Pediatric Section of Fulton County Medical Society, Atlanta, Ga.

Chairman: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Secretary: Dr. William Kiser, Medical Arts Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St. Time: Second Thursday of each month from October to April, 8 p. m.

New England Pediatric Society

President: Dr. Edwin T. Wyman, 483 Beacon St., Boston.
Secretary-Treasurer: Dr. Randolph K. Byers, 66 Commonwealth Ave., Boston.
Place: Boston Medical Library. Time: Second Friday of each month.

Chicago Pediatric Society

President: Dr. Arthur H. Parmelee, 310 S. Michigan Blvd., Chicago.
Secretary: Dr. Bert I. Beverly, 130 S. Oak Park Ave., Oak Park, Ill.
Place: City Club. Time: Third Tuesday of each month.

Louisiana State Pediatric Society

President: Dr. J. A. Crawford, Lake Charles.
Secretary-Treasurer: Dr. C. T. Williams, 7401 Burthe St., New Orleans.

Detroit Pediatric Society

President: Dr. Wyman C. C. Cole, 2970 W. Grand Blvd., Detroit.
Secretary: Dr. Charles A. Wilson, 71 E. Perry St., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

University of Michigan Pediatric and Infectious Disease Society

President: Dr. Paul W. Beaven, 26 S. Goodman St., Rochester, N. Y.
Secretary: Dr. John P. Parson, 320 S. Division St., Ann Arbor, Mich.
Place: University Hospital, Ann Arbor, Mich. Time: Nov. 8-9, 1929.

Northwestern Pediatric Society

President: Dr. C. R. Moriarity, La Salle Bldg., Minneapolis.
Secretary: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Place: University Campus and Rochester, Minn. Time: Winter, Spring, Summer, Fall.

St. Louis Pediatric Society

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary-Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

Nebraska-Iowa Pediatric Society

President: Dr. H. B. Hamilton, 434 Aquila Court Bldg., Omaha.
Secretary: Dr. George L. Clark, 1817 Vinton St., Omaha.
Place: As announced by committee. Time: Second Thursday of each month from October to June, inclusive, 6 p. m.

Brooklyn Pediatric Society

President: Dr. Louis M. Ruderman, 961 Eastern Pkwy., Brooklyn.
Secretary: Dr. Joseph F. Paulonis, 1357 Buschwick Ave., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

Bronx Pediatric Society

President: Dr. L. H. Barenberg, 1749 Grand Concourse, New York.
Secretary: Dr. Joseph Golomb, 496 E. 175th St., New York.
Place: Concourse Plaza, 161st St. and Grand Concourse. Time: Second Wednesday of each month, except June, July, August and September.

Central New York Pediatric Club

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Place: Various cities in New York. Time: Fourth Thursday of October, January and April.

New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. F. Elmer Johnson, 300 West End Ave., New York.
Secretary: Dr. Hugh Chaplin, 39 E. 75th St., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

Rochester Pediatric Society

President: Dr. John Aikman, 184 Alexander St., Rochester, N. Y.
Secretary: Dr. Howard F. Rowley, 278 Alexander St., Rochester, N. Y.
Place: Arranged by program committee. Time: First Friday of each month from October to May.

Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. C. W. Wyckoff, 8314 Euclid Ave., Cleveland.
Secretary: Dr. C. L. Ruggles, 10616 Euclid Ave., Cleveland.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

Oklahoma State Pediatric Society

President: Dr. William M. Taylor, Osler Bldg., Oklahoma City.
Secretary: Dr. Carol Pounders, 110 W. 10th St., Oklahoma City.
Place: Shawnee. Time: May, 1930.

Philadelphia Pediatric Society

President: Dr. Ralph M. Tyson, 334 S. 21st St., Philadelphia.
Secretary: Dr. John D. Donnelly, 115 Bryn Mawr Ave., Bala-Cynwyd, Pa.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday of each month from October to June, inclusive.

Pittsburgh Pediatric Society

President: Dr. Zaccheus R. Scott, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. Theodore C. Elterich, Highland Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate months.

The South Carolina Pediatric Society

President: Dr. E. A. Hines, Seneca.
Secretary-Treasurer: Dr. R. M. Pollitzer, 105 E. North St., Greenville.

Memphis Pediatric Society

President: Dr. A. G. Quinn, 1193 Madison Ave., Memphis, Tenn.
Secretary: Dr. Kinsey M. Buck, 1024 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

Texas Pediatric Society

President: Dr. H. P. Ledford, 1310 9th St., Wichita.
Secretary-Treasurer: Dr. John G. Young, 3930 McKinney Ave., Dallas.

Richmond Pediatric Society

President: Dr. J. B. Stone, 2042 Park Ave., Medical Arts Bldg., Richmond, Va.
Secretary-Treasurer: Dr. Samuel A. Anderson, Jr., 2326 W. Grace St., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St. Time: First Thursday of each month (other than during the summer).

Virginia Pediatric Society

President: Dr. Franklin D. Wilson, 136 York St., Norfolk.
Secretary: Dr. J. B. Stone, 2042 Park Ave., Richmond.

North Pacific Pediatric Society

President: Dr. W. F. Patrick, Medical Arts Bldg., Portland, Ore.
Secretary: Dr. L. Howard Smith, Medical Arts Bldg., Portland, Ore.

Seattle Pediatric Society

President: Dr. Vernon W. Spickard, Cobb Bldg., Seattle.
Secretary: Dr. Raymond H. Somers, Cobb Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, 7 p. m.

Milwaukee Pediatric Society

President: Dr. A. L. Kastner, 120 E. Wisconsin St., Milwaukee.
Secretary: Dr. F. R. Janney, 418 E. North Ave., Milwaukee.
Place: Children's Hospital. Time: Second Wednesday of each alternate month, beginning with February.

Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. G. A. Davis, 12 Belsize Drive, Toronto.
Secretary: Dr. Gladys Boyd, 56 DeLisle St., Toronto.
Time: November 17, December 15, February 16 and March 15.

INDEX TO VOLUME 38

The star (*) preceding the page number indicates an original article. No author entries are made except for original articles. Biographies, Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O and S, respectively.

PAGE

Abdomen, abdominal pain in children.....1326
acute, in early life, from physician's standpoint 418
Abnormalities: See also Monsters; Twins
ami logenous malformation of skull and brain 848
anomalies of urinary tract in infants....*1202
arachnodactylia 608, 1296
congenital atresia of aortic ring..... 156
congenital bilateral microophthalmos accompanied by other malformations..... 185
congenital defects of radius and ear and facial paralysis 161
congenital deformity of arm (hemielectromelial) with total absence of hand and rudimentary radius and ulna..... 608
congenital one-sided defect of kidney with presentation of 3 new cases of agenesia and 1 of aplasia..... 156
congenital structural anomalies of pulmonary artery 158
congenital synostosis of maxilla and mandibula 609
duplex jejunum 1079
ectopia cordis pectoralis.....1296
ectopia cordis pectoralis; electrocardiographic study 1297
hereditary ectodermal dysplasia.....1296
hereditary symmetrical hypoplasia of joints 161
macrosomia adiposa, familial congenital.. 849
malformation of left ventricle..... 848
malformation of organs of respiration.... 848
malformations of extrahepatic bile ducts 849
megalosyndactilitis; report of case..... 863
radium therapy of uterine cancer during pregnancy followed by microcephalus..1102
thoracopagi 1296
tricuspid valve, rare malformation of.. 848
tumor-like nodule in lateral wall of spinal cord in human embryo of 16.5 cubic millimeters 159
unilateral hypertelorism 416
Abscess, cerebral, report of case..... 875
cold, rupture of, in Pott's disease into pleuro-pulmonary passages and into digestive tract 1304
lung, experimental production of....669, *683
paranephric 887
perirenal, in infancy..... 419
peritonsillar, in infant aged 3 weeks.... 892
retropharyngeal, in infants and children 638
rupture of heart from..... 858
Acetaldehyde, meta poisoning in childhood 889
Acetarsone therapy for trichocephalus dispar 1099
Acid, effect of fruit juices and, on behavior of excised intestines..... 601
Acid Base Equilibrium, alterations in acid base metabolism in alimentary and in sodium chloride fever..... 403
Acidosis, method of restoration of body fluids 658
of acute diarrhea in infancy.....*314
of cyclic vomiting, influence of emotional shock and rôle of nervous system in..1327
Acids, nucleic: See Nucleins
Acrodynia, case report.....1111
infantile 1107
Swift-Feer, its relationship to epidemic neuraxitis 173
Acterol: See Ergosterol, irradiated
Adenitis: See under Lymph Nodes
Adenoidectomy, effect of tonsillectomy and, on diphtheria immunity.....432, *929

PAGE

Adenoids and pharyngeal hypophysis..... 193
Agglutinins, Brucella abortus in milk as source of, in human sera.....1073
icterus neonatorum as consequence of iso-agglutination 1077
immune, inheritance of agglutinogens of human blood demonstrable by..... 157
iso-agglutinins in breast milk.....1292
Agnesia: See under Abnormalities
Agranulocytosis: See Angina, Agranulocytic
Alastrim: See Amaas
Aleurons and protein cereals.....1290
Alkalosis, arsenical reactions in congenital syphilis complicated with..... 623
method for restoration of body fluids..... 658
Allergy: See Anaphylaxis and Allergy
Alopecia areata, etiology of..... 640
Amaas, cases of alastrim in Rotterdam....1086
Amberg, S.: Paroxysmal tachycardia with syncope occurring in a child.....*551
Amino-Acids in blood, determination of; note on Folin's method; amino-acids in health and disease.....1286
influence of, on cultures of diphtheria bacilli 618
Amnion, adhesions of..... 847
Amyotonia: See Myatonia
Anaphylaxis and Allergy; allergy and immunity in trichophyton infections.....1322
allergy in children..... 171
changes in serum cholesterol in..... 599
idiocyncrasy to egg; rôle of allergy in some dermatoses especially eczema....1322
infantile pyloric obstruction; preliminary report of its allergic nature.....*741
influence of cholesterol on experimental anaphylaxis 844
relation of scarlet fever to allergy..... 613
serum anaphylaxis, researches on..... 149
Anemia, aplastic, report of case..... 219
Gaucher's disease in infants, anatomy of 865
Gaucher's disease; review of literature and report of case diagnosed from section of inguinal gland.....*775
liver therapy of anemias.....633, 1097
nutritional, in infancy, observations on a common deficiency disease..... 415
pernicious and aplastic, and leukemias, borderline cases 632
pernicious and aplastic, in infancy..... 860
pernicious, in children..... 174
sickle cell, in a Greek family.....*103
sickle-cell, with report of splenectomy...1323
Anesthesia in children.....1337
Aneurysm, congenital, of cerebral arteries. 157
Angina, first case of agranulocytosis reported in Japan.....*1037
pathogenesis of sepsis after; histologic observations 1106
Plaut Vincent's, etiology and etiological treatment of..... 191
Anhydremia: See Blood, water content
Anomalies: See under Abnormalities
Anorexia: See Appetite
Anthrax, pneumonic, in a child.....*1252
Anthropometry, height-weight-age table for Chinese children 1110
physical traits of young children; report of measurements of 150 boys and 167 girls from 3 to 6 years of age.....*541
relations of weight, height and age in negro children *1222
school 1328
Antigens and Antibodies, effect of ultraviolet irradiation on formation of..... 601
in mother and fetus..... 849
Antrum, latent disease of maxillary sinus.. 192
Anus, imperforate, with exit through prostatic urethra; report of 3 cases.....*559
Aorta in children.....1091
Aplasia: See under Abnormalities
Appendicitis, acute 897
clinical aspects of, in children..... 675

PAGE

Appendix, fibroplastic inflammation of cecum, ascending colon and.....1312
function of 400
Appetite, psychoanalysis applied to, in children 425
Arachnodactylia: See Abnormalities
Armand-Delille, P.: Diagnosis of tuberculosis of tracheobronchial glands.....*1125
Arrhythmia, auricular fibrillation..... 674
Arsphenamine, intravenous injections of neoarsphenamine for chorea minor..... 427
Arteries, cerebral, congenital aneurysm of 157
pulmonary, congenital structural anomalies of..... 158
rôle of arterial occlusion and anoxemia in cause of cerebral diplegias..... 423
Arthritis, gonorrheal, in new-born.....1077
Ascaris lumbricoides and related heminths, precipitin reactions with various tissues of 144
lumbricoides, sudden death due to occlusion of larynx by..... 192
Ascites, hepatic cirrhosis and splenomegaly with; complete relief from ascites under antisyphilitic treatment..... 624
Askin, J. A.: Encephalitis accompanying whooping cough; clinical history and report of postmortem examination..... *97
Asthma-eczema-prurigo complex 198, 199
bronchial, in early childhood.....1310
natural history of.....1325
Ataxia, cerebellar, acute..... 179
Atrophy, of subcutaneous fat, following injection of insulin; frequency of.....*715
August, M. H.: Studies in scarlet fever; blanching with placental serum.....*953
Auricular Fibrillation: See Arrhythmia

B. C. G.: See under Tuberculosis
Baciluria: See under Urine
Bacillus: See Bacteria
Bacteremia and skin manifestations in lipoid nephrosis *762
Bacteria: See also Diphtheria Bacilli; Tubercle Bacilli; etc.
Brucella abortus in milk as source of agglutinins in human sera.....1073
Brucella abortus in milk, thermal death point of 597
Calmette-Guérin bacillus: See under Tuberculosis
colon bacillus antiserum of Vincent, infection of urinary tract in infants treated with 858
destruction of diphtheria toxin by..... 147
passage of living bacteria through intestinal wall 143
Barbital and Barbital Derivatives; phenylethylhydantoin in chorea.....876, 1317
phenylethylhydantoin, results of therapy. 876
Beal, F.: Preparation of dextrose for intraperitoneal injection *943
Behavior and constipation.....*570
difficulties in children associated with results of birth trauma..... 634
Beinhauer, L. G.: Purpura hemorrhagica gangrenosa *1013
Benzene, involution and regeneration of thymus under influence of..... 844
Benzidine Test: See Urine
Beriberi in infants..... *52
infantile, with symptoms of human milk intoxication 156
Bizier, J. A.: Anomalies of urinary tract in children; frequency and types in a consecutive necropsy series.....*960
Interpretation of roentgenograms of chest in children based on necropsies; hilar shadows and linear markings.....*978
Interpretation of roentgenograms of chest in children based on observations at necropsy *1166
Bile Ducts, common, effect of ligation of, upon the appearance of tetany in thyroparathyroidectomized dogs 144
congenital atresia and stenosis of..... 607
extrahepatic, malformations of..... 849

PAGE

Biographies:
Harris, Walter, 1647-1742.....*1064
Hoefer, Wolfgang, 1614-1681..... *834
Mayow, John, 1643-1679..... *590
Starsmare, J., 1664.....*1277
Sydenham, Thomas, 1624-1689..... *393
Wiseman, Richard, 1622-1676..... *137
Birnberg, T. L.: Cardiospasm in new-born infant, report of 3 cases.....*1183
Birth injuries, can they be prevented?.... 849
rate and infant mortality in Bavaria.... 890
trauma, behavior difficulties in children associated with results of..... 634
trauma, cerebral injury at birth with maldevelopment and myxedema.....1334
Blackader, Alexander D., lectureship in honor of.....1281
Bladder, exstrophy of; 2 cases..... 159
fistula with osteomyelitis.....1334
malacoplakia of, etiology..... 865
roentgenogram of child with chronic pyuria 1101
Blank, G. M.: Unusual association of postinfantile rickets with osteomalacia..... *84
Blepharitis: See under Eyelids
Blind and deaf girl, individual education of 1109
Blood: See also Hemoglobin; Leukocytes
amino-acids in, determination of; note on Folin's method; amino-acids in health and disease.....1286
ammonia in, pathologic significance of increase during convulsions in dysentery 620
calcium and inorganic phosphorus in; influence of light environment on normal rabbits 597
cells, study on living blood cells of newborn 1297
chemical blood analysis and its value to hospital 1331
chemical changes in, during fasting and subsequent refeeding; inorganic components 1285
chlorine and iron in, accurate microdetermination of.....1074
chlorine content of whole blood in eczema 883
cholesterol content of, in hydrolabile children 149
cholesterol in diabetic children.....*490
cholesterol of healthy and sick children.1285
cholesterol of plasma in epilepsy.....1313
cultures after tonsillectomy.....*726, *1282
differential count of white corpuscles in stained blood smears, source of error in 151
examination in new-born and treatment for hemorrhage 1075
groups and paternity 201
groups, inheritance of 201
groups, isohemagglutination in mothers and new-born children 852
in tuberculosis in childhood..... 170
permeability of cerebrospinal fluid and, of infants for bromine..... 599
platelets, persistence of hematologic signs after clinical cure in syndrome of Werlhof 859
platelets, rate of disintegration in normal children, in hemophilia and in thrombopenia 600
polymorphonuclear count in new-born...*271
polymorphonuclear count in new-born, deflection by ultraviolet light.....*547
pressure and exudative diathesis..... 420
pressure, malignant hypertension.....1333
protein content of plasma in infantile eczema 642
protein content of serum, in eczematous infants 428
protein in childhood..... 150
reticulocytes in embryos and the newborn 851
sedimentation test in experimental poliomyelitis 841
serum phosphates in toxicosis..... 599
serum, pooled adults, as prophylactic measure in measles 219
sickle-shaped red cells, origin and fate of 399
sugar curve and glucose tolerance.....1074
sugar during fasting; carbohydrate metabolism of premature infants.....*912

Blood—Continued PAGE
sugar, spontaneous hypoglycemia in childhood *746
sugar studies in endocrine dysfunctions using author's micro-Folin-Wu method of estimation 1095
sulphur in, of children.... 600
transfusion and vaccine therapy in diseases of children....1327
transfusion, etiology and pathologic anatomy of reactions after.... 152
transfusion in children....1333
transfusion, intraperitoneal 1328
transfusion, rôle of, in pediatric practice 433
transfusion, thrombocytopenia - purpura treated by splenectomy and repeated... 672
values in new-born infant, relationship of jaundice and weight to....*518
volume and diuresis due to salyrgan....1098
water contents of; alterations in acid base metabolism in alimentary and in sodium chloride fever.... 403
Body fluids, improved method for restoration of 658
process of adipolysis in course of loss of weight in infancy.... 604
relationship of jaundice and weight to blood values in new-born infant....*518
weight abnormal, basal metabolism in children of *299
weight and sitting height, in new-born infants; studies of.... 610
weight, height and age, relation between, in negro children....*1222
weight, increase in, of healthy children during first year....1293
weight, range table, for children from 5 to 15 years of age....*758
Bonar, B. E.: Bilateral congenital facial paralysis; review of literature on congenital facial paralysis and a classification *1256
Bone changes in continuous phosphorus ingestion 1344
composition, effect of massive doses of irradiated ergosterol on.... 442
destructive changes in bony shafts in congenital syphilis 1305
development of 1331
diseases in infancy and childhood.... 414
diseases in syphilis....1092
dystrophy with characteristics of leontiasis ossea, osteitis deformans and osteitis fibrosa cystica in child; influence of central nervous system....*348
fragilitas ossium treated with thymus preparations 1100
growth; diabetic exophthalmic dysostosis. 627
growth; familial pleonosteosis.... 173
growth; statistical study of rate of skeletal growth in juvenile diabetes....1095
growth, stimulation by injections of extract of bone.... 600
properties of chondroid zone of rachitic bones....1290
Book Reviews:
Applied Electrocardiography; A. E. Parsonnet and A. S. Hyman....1348
Biochemistry of the Amino Acids; H. H. Mitchell and T. S. Hamilton.... 222
Blood Picture; V. Schilling. Translated and Edited by R. B. H. Gradwohl....1348
Care and Feeding of Children; L. E. Holt, Jr. 1120
Child of Circumstances; A. Wilson.... 677
Child's Heredity; P. Popenoe....1120
Diagnostik und Therapie der Kinderkrankheiten; F. Lust.... 900
Diseases and Deformities of the Spine and Thorax; A. Steindler.... 445
Diseases of Women: Symptoms and Treatment; F. I. Schroyer....1348
Fuel of Life; J. J. R. Macleod.... 222
Helminthes et protozoaires les plus frequents; Leo 899
Klinische Gasstoffwechseltechnik; H. W. Knipping and H. L. Kowitz.... 678
Mental Deficiency (Amentia); A. F. Tredgold 899

Book Reviews—Continued PAGE
Modern Methods of Feeding in Infancy and Childhood; D. Paterson and J. F. Smith 1349
New-born Infant; E. L. Stone.... 446
Notes on Chronic Otorrhoea; A. R. Friel 446
Nutrition of Healthy and Sick Infants and Children; Nobel, Pirquet and Wagner. Translated by B. M. Gasul....1349
Osteomyelitis and Compound Fractures; H. Winnett Orr.... 677
Physical Measures of Growth and Nutrition; R. Franzen....1349
Rickets, Including Osteomalacia and Tetany; A. F. Hess....678
Selected Articles. From the Department of Pediatrics, University of Toronto, and Hospital for Sick Children, Toronto.... 900
Stations thermales du centre de la France; P. Carnot 446
Study of Masturbation and the Psychosexual Life; J. F. W. Meagher....1120
Therapeutics Vademecum für die Kinderpraxis; H. Kleinschmidt.... 900
Three Minute Medicine; L. R. Effler.....1350
Boorstein, S. W.: Congenital backward dislocation of knee....*107
Boric Acid, time of absorption and excretion of, in man.... 145
Boyd, G. L.: Blood cholesterol in diabetic children *490
Boyd, J. D.: Dietary control of dental caries *721
Brain, birth injury of.... 162
cephalic metastases of suprarenal blastomata in children.... 172
congenital encephalopathy and heredosyphilitic 876
hemorrhage in new-born infant as demonstrated by roentgen rays....*1196
injury at birth with maldevelopment and myxedema 1334
intracerebral calcifications in childhood.. 426
lead encephalopathy, neuropathologic changes in case of.... 870
localization of vegetative centers in cerebral cortex 1283
thymogenous capillary atony as cause of cerebral disturbance.... 182
tumors, in childhood; clinicopathologic study 869
tumors of infancy 180
tumors of posterior fossa, encephalography of 184
vitamin content of brain tissue compared with vitamin A in retinal tissue.....1074
Breast-Feeding: See Infant Feeding
Breast Milk: See Milk, human
Breithart, J.: Infantile pyloric obstruction; preliminary report of its allergic nature *741
Bromine, permeability of blood and cerebrospinal fluid of infants for.... 599
Bronchiectasis, chronic, in childhood, early diagnosis of 627
treatment of 1095
Bronchitis, laryngotracheobronchitis, acute. 628
Broncho-pneumonia 858, 1073
and croupous pneumonia.... 628
and whooping cough, generalized emphysema in 616
roentgen examination in; pathogenesis and clinical course....1098
tuberculous, in an infant.... 170
Bronchus, complete rupture of right bronchus 865
pipe stem in....1105
Brucella Abortus: See under Bacteria
Bulbocapnine in juvenile behavior disorders in epidemic encephalitis.... 874
Bursa, cystic hygroma in a Mongol..... 416
Burt, E. F.: Continuous temperature records of premature infants.....*944
Buttermilk: See under Milk

Caffey, J.: Kahn precipitation test in infancy and early childhood; a comparative clinical study with Noguchi-Wassermann reaction *1206
Calcification, calcinosis and scleroderma in child treated by ketogenic diet..... 603
intracerebral, in childhood..... 426

Calcinosis: See Calcification PAGE
Calcium, ash content of growing organism.. 151
calcium-soap stools 1293
metabolism as related to therapy..... 202
potassium and magnesium contents of spinal fluid 1320
Campbell, M. F.: Suppurative epididymitis in infancy *794
Cancer in 3 year old child..... 175
of larynx in young..... 428
Capillaries, thymogenous atony as cause of cerebral disturbances 182
Caput Obstipum: See Torticollis
Carbohydrate content of foods..... 603
experiments in feeding low carbohydrates 155
metabolism changes in cirrhotic change of liver 1092
metabolism in progressive muscular dystrophy 1315
metabolism of premature infants; blood sugar during fasting.....*912
tolerance in infants and in young children with celiac disease..... *16
Carbon Arc: See Ultraviolet Rays
Cardiospasm: See Stomach
Caries, circular of deciduous teeth, pathogenesis of 1309
dental, malformations and.....1337
Cataract, congenital, and changes in neurovegetative apparatus, relation between. 190
juvenile, endocrine etiology of 191
juvenile, spontaneous resorption of 1104
tetany and dwarfism..... 190
Cecum, fibroplastic inflammation of appendix, ascending colon and.....1312
Celiac Disease, carbohydrate tolerance in infants and in young children with.... *16
two cases of celiac infantilism.....1309
Cerebrospinal Fluid, anatomy of subarachnoid space and barrier between blood and spinal fluid.....1284
chemistry of 638
lactic acid content and *p*H of, in tuberculous meningitis 414
lactic acid of, as a prognostic and therapeutic guide in meningitis..... 211
permeability of blood and, of infants for bromine 599
potassium, calcium and magnesium contents of 1320
protein content in myxedema..... 145
refractive index in normal and pathologic conditions 875
relation of streptococci to, in experimental poliomyelitis 596
temporary loss of eyesight due to pressure of 185
Cesium eosinate in urticaria and allied disorders 200
Character building, influence of home and institutional training on..... 425
Chen, K. T.: Typhoid fever; report of case in infant less than 1 month of age...*1044
Chest: See Thorax
Chickenpox and herpes zoster.....428, 1299
and herpes zoster, statistical study.....1299
and whooping cough, prevention and treatment 407
complicated by acute nephritis; associated with streptococcal infection of tonsils.. 825
immunization against 855
value of von Kézmárszky's method of immunization against 616
Child Welfare, bibliographic information for workers 201
Brazilian council for assistance and protection of minors..... 890
children's health consultation in New York 1071
defective children of primary schools in province of Buenos Aires..... 889
in Holland 140
influence of home and institutional training on 425
institutions for care of children in Woj. Kieleckie 201
Maryland school children examined.....1071
Child Welfare—Continued PAGE
maternity and child welfare with reference to legislation..... 202
medical aspects of nursery school.....1337
pedologic advisory clinic in Warsaw.... 201
President Hoover calls conference on child health 593
scholastic efficiency of school children without full complement of parents....1109
serving the child in Fargo.....1324
the individual and the sphere of work for the good of society..... 201
Chinese children, height-weight-age table for 1110
Chlorine in blood and other liquids, accurate micro-determination of.....1074
Cholera, vitality of vibrio cholera on Chinese paper money.....1088
Cholesterol, changes in serum cholesterol in anaphylaxis 599
in blood of healthy and sick children; its biologic action 1285
influence on experimental anaphylaxis... 844
of blood plasma in epilepsy.....1313
Chondrodysplasia 1334
Chorea, chronic 1338
Huntington's, symptomatology of different stages of 1314
levulose test in.....1081
minor 636
minor, intravenous injections of neoarsphenamine for 427
pathogenesis of involuntary movements with reference to 1315
phenylethylhydantoin in 876, 1317
Chvostek's Sign: See Tetany
Circumcision, infant 644
Cisterna Magna, puncture of..... 634
Cod Liver Oil and activated ergosterol therapy 603
for reproduction 144
therapy 155
Cohen, M B.: Infantile pyloric obstruction; preliminary report of its allergic nature *741
Colds: See under Respiratory Tract
Colic, relation between eczema and, in early infancy 660, *935
Colitis, mucous, roentgenologic sign of.... 627
Collens, W. S.: Influence of ultraviolet rays, yeast and irradiated yeast on the sugar tolerance of diabetic children...*513
Management of ambulatory diabetic child.*275
Colon, ascending, fibroplastic inflammation of appendix, cecum and.....1312
dilatation (megacolon) 1096
Hirschsprung's syndrome as first sign of congenital myxedema 861
Color Blindness, frequency of congenital anomalies of color sense in boys and girls 190
Communicable Diseases; alterations in myocardium in infectious diseases.....1310
can quarantine regulations for diphtheria and scarlet fever be less stringent than for measles and pertussis.....1300
communication and control of.....1080
discussion on contagious diseases..... 408
inheritability of susceptibility to infectious diseases 618
Conjunctiva, bulbar, nevus of..... 186
hemorrhage from, in infant following instillation of silver nitrate solution..... 186
palpebral, mycosis of..... 189
Conjunctivitis, staphylococcus aureus, of new-born 161
Constipation and behavior.....*570
treatment by use of residue-free diet.... 444
Contagious Diseases: See Communicable Diseases
Convulsions in new-born infant..... 610
Cooley, T. B.: Sickle cell anemia in a Greek family *105
Cooperstock, M.: Comparative effects of diphtheria toxoid and toxin-antitoxin as immunizing agents *35
Cornea, bilateral cysts of..... 188

PAGE

Correction to "Multiple disseminated lupus vulgaris" by V. C. Garner [American Journal Diseases of Children 38: 1036 (November) 1929] 1255
Costa's Reaction: See Tuberculosis
Cowpox vaccine, localization of, in blood of vaccinated guinea-pig 599
Cranium, craniosynostosis (oxycephaly and related disorders) 157
increased intracranial pressure..... 634
intracranial hematoma in new-born.....1078
ocular troubles in certain cases of tumors of anterior floor of..... 874
Creatine and Creatinine; effect of high and low protein diets on excretion of creatinine, ammonia and creatine.....*468
growth of infants from the standpoint of physical measurements and of nitrogen metabolism 499, *507
Crying, incessant, in young children..... 648
Curtin, V. T.: Progeria; review of literature with case report.....*993
Cutler, E. C.: Experimental production of abscess of lung.....*683
Cyanosis, congenital (morbus cerulcus).... 418
of face and arms in newly born..... 851
Cyst, arterial supply of congenital polycystic kidney 145
formation in lungs.....1311
of liver, solitary nonparasitic.....*241
of tongue in congenital stridor..... 428
Cystogram: See under Bladder.....1101

Daniels, A. L.: Growth in infants from standpoint of physical measurements and nitrogen metabolism; uric acid....*507
Growth of infants from standpoint of physical measurements and of nitrogen metabolism; creatine *499
Davis, E.: Ureteral neuromuscular dystrophy in relation to infections of kidneys *1006
Deaf and blind girl, individual education of.1109
Death Rate: See Infant Mortality; Vital Statistics
Defectives, games of defective children....1110
Deformities: See under Abnormalities
Dehydration 666
De La Pena, A.: Horseshoe kidney.....*799
Dentition: See under Teeth
Dermatomyositis 429
De Sanctis, A. G.: Determination of value of serum in meningococcus meningitis; report of illustrative series of cases...*730
Dextrocardia: See Heart
Dextrose, apparatus for administering solution of 675
blood sugar curve and glucose tolerance.1074
preparation for intra-peritoneal use.....*943
Diabetes Insipidus, antidiuretic effect of solution of pituitary dropped intranasally in *342
case report 1335
discussion of question of polydipsia..... 868
experimental 842
the diuretic substance 841
Diabetes Mellitus, blood cholesterol in diabetic children *490
in a mongolian imbecile 874
in children 1094
influence of ultraviolet rays, yeast and irradiated yeast on sugar tolerance of diabetic children *513
juvenile, management of.....1311
juvenile, statistical study of rate of skeletal growth in.....1095
juvenile, treatment of.....*323
juvenile, treatment with synthalin..... 863
management of ambulatory diabetic child.*275
nutrition of diabetic children.....1293
sorbite as source of carbohydrate in.... 420
treatment with low fat diet..... 865
Diaphragm, saucer deformity of, and its origin 1102
Diarrhea, acid, treatment by adsorbents... 605
acute, in infancy, acidosis of.....*314
conditions in the southern states..... 401
Diathesis, exudative, and blood pressure.. 420
exudative, and hemotherapy..... 631

PAGE

Dick, G. F.: Control of scarlet fever.....*905
Dick, G. H.: Control of scarlet fever.....*905
Dick Reaction and smallpox vaccination... 620
Dickey, L. B.: Size of reacting area in intracutaneous tuberculin tests in relation to classification of disease and to other clinical factors.....*1155
Diet: See also Nutrition
and dental caries.....439, *721
antenatal and postnatal, effect on structure of teeth and incidence of dental caries 154
effect of rickets and severe nutritional disturbances on children's teeth..... 440
high and low protein, effect of, on excretion of creatine, creatinine and ammonia 468
ketogenic, in calcinosis and scleroderma in child 603
low fat, in treatment of diabetes..... 865
residue-free, in treatment of constipation 444
Digitalis, studies on children with heart disease *699
therapy, its uses in children..... 415
Diphtheria 1082
active immunization against, results in Berlin 1300
active immunization with toxoid.617, 1084, 1332
antidiphtheritic vaccination with anatoxin.1081
bacilli and ragweed pollen, hypersensitiveness to 843
bacilli, hypersensitiveness to.....613, 842
bacilli in male urethra..... 410
bacilli, influence of certain amino-acids on cultures of..... 618
bacilli, passive transfer of hypersensitiveness to 842
carriers, investigations of..... 409
electrocardiographic changes in.....1079
extensive, diffuse, hydropic degeneration of myocardium associated with diphtheritic infection 612
fermentative method of producing toxin in vitro 618
hemiplegia after 408
immunity, effect of tonsillectomy and adenoidectomy on 432, *929
immunization, in Philadelphia.....1071
in South Carolina, study of 100 deaths from, during the years 1926-1927..... 853
negative Schick reaction in new-born and nursing infants 1082
1895-1914 1079
percutaneous immunization according to Loewenstein 1085
postdiphtheric stenosis of cicatrices of larynx in children and their treatment 164
present treatment for.....1081
prevalence of, and susceptibility to, in North Carolina 1080
protective action of serum against hemolysin in 149
serum resistance in.....1081
statistical study of scarlet fever and.... 613
susceptibility of Eskimos to common cold and study of their natural immunity to 163
toxin, destruction of, by bacteria..... 147
toxin, in vitro, approximation of toxicity of 163
toxin, influence of lipoids on action of, in animal experiments..... 411
toxin, study of certain effects occasioned in dogs by..... 399
toxin-antitoxin, effect of low temperatures on 597
toxoid and toxin-antitoxin as immunizing agents *35
tracheal foreign body simulating..... *95
vegetative nervous system in.....1087
Diplegia: See Paralysis
Diplopia, functional, in school boy..... 880
Diuresis due to salyrgan and blood volume.1098
Diverticulum, congenital, of esophagus.... 607
*Drain, C. L.: Dietary control of dental caries *721*
Duodenum, congenital valves, pseudovalves and stenosis of pyorus and..... 846

PAGE

Dwarfism, cataract and tetany..... 190
review of literature of progeria and case report *993
Dysentery, bacillary 1082
bacillary and amebic, differentiation by cytodiagnosis 1088
pathologic significance of increase in blood ammonia during convulsions in.. 620
Dysostosis: See Bone, growth
Dysplasia, hereditary ectodermal.....1296
Dystrophy, hereditary ectodermal.....1321
local fat, after injection of insulin..... 420
muscular, progressive and carbohydrate metabolism in 1315
muscular, pseudohypertrophic 674
osseous, familial 417
progressive muscular, a form not described in literature..... 876

Ear, acute discharging, indications for operative interference 881
as erotic organ in children..... 426
congenital arteriovenous fistula in tympanum 1105
congenital aural fistula.....1339
infections, in children..... 639
middle, acute suppurations, acute mastoiditis and their complications; bacteriology and clinical course of..... 197
middle, infections of upper respiratory tract and 856
middle, primary tuberculosis of, in infants 193
Ectodermal Defect, congenital..... 833
hereditary ectodermal dysplasia.....1296
hereditary ectodermal dystrophy.....1321
Ectopia Cordis: See under Heart
Eczema 660
asthma-prurigo complex 198, 199
chlorine content of whole blood in..... 883
infantile, protein content of plasma in.. 642
infantile, protein content of blood serum of 428
infantile, water metabolism in..... 642
marginatum in a nursling.....1323
relation between colic and, in early infancy 660, *935
rôle of allergy and idiosyncrasy in.....1322
Edema, angioneurotic 897
nutritional 1291
unusual case of.....1336
Egg, idiosyncrasy to: See under Allergy
white, thread-forming property and structure in 400
Eisendrath, D. N.: Ureteral neuromuscular dystrophy in relation to infections of kidneys *1006
Electrocardiograms, effect of digitalis on, of children with rheumatic fever and chronic valvular heart disease.....*699
in dextrocardia *112
Embolus of central retinal artery.....1321
Emesis: See Vomiting
Emphysema, generalized, in bronchopneumonia and whooping cough..... 616
Empyema 203
aspiration in 203
conservative methods of dealing with.... 200
in childhood 204
in childhood, types of pneumococcus in.. 175
pneumococcic, intrapleural injections of optochine (ethyl hydrocuprein hydrochloride) for 220
pneumococcus, of early childhood; conservative treatment 887
pneumonia, nephritis and pneumococcal peritonitis with complete recovery....1309
staphylococcic, in infant.....1332
Encephalitis: See also Encephalomyelitis; Meningo-Encephalitis
accompanying whooping cough; clinical history and report of postmortem examination *97
chronic epidemic, conjugate ocular muscle spasm in 877
chronic epidemic, oculogyric crises in... 424
complicating pneumonia 1103
constitution, predisposition and heredity in, with special consideration of constitution and racial factors..... 180

Encephalitis—Continued PAGE

following pertussis 1340
in origin of mental defects.....1103
in pertussis 615
juvenile behavior disorders in epidemic encephalitis, bulbocapnine in..... 874
periaxalis diffusa 634
periaxialis diffusa of Schilder.....423, 870
postencephalitic tremor 179
postvaccinal 184, 411, 1102
pregnancy, labor and puerperium in epidemic encephalitis 1075
respiratory sequelae 176
Swift-Feer acrodynia, its relation to epidemic neuraxitis 173
Encephalography in tumors of posterior fossa 184
Encephalomyelitis in virus diseases and exanthemata 178
in virus infections..... 178
postvaccinal 855
Encephalopathy: See under Brain
Endarteritis calcificans cerebri and cerebral calcification epilepsy 422
obliterans, extensive visceral, in child....1308
Endocarditis, subacute bacterial, occurrence of, in childhood..... 630
Endocrine dysfunctions; blood sugar studies in, using author's micro-Folin-Wu method of quantitative estimation.....1095
Enteric Fever: See Typhoid
Enzymes, digestive, of human fetus..... 399
Epididymitis, suppurative, in infancy.....*794
Epilepsy and allied conditions in children.. 877
cerebral calcification, and endarteritis calcificans cerebri 422
cholesterol of blood plasma in.....1313
in children *1068
infections as a source of recurrent epileptiform seizures 1325
intelligence of epileptic children.....1325
laboratory findings in..... 871
relationship of water balance to the occurrence of seizures.....*451, 667
treatment for manifestations by phenylethyl manonylurea 179
Epinephrine, sensitiveness of children with measles and scarlet fever to cutaneous injections of 616
Epiphysiolysis of femoral head at birth...1109
Epstein, I. M.: Blood cultures after tonsillectomy *726
Ergosterol, activated therapy and cod liver oil 603
in manifest spasmophilia 637
irradiated, a method of assay of antirachitic vitamin D..... 1287
irradiated, value of acterol (irradiated cholesterol) in treatment of parathyroidectomized dogs..... 145
irradiated, action of, in rabbit..... 399
irradiated, effect of massive doses on composition of bone..... 442
irradiated, experiences with 431
irradiated, in tetany 422
irradiated, or irradiated milk 606
irradiated, researches on 890
irradiated, severe damage to rabbits through 600
irradiated, toxicity of 401
irradiated, treatment and prevention of rickets with 402
Erythema nodosum 891
nodosum and tuberculosis.....1107
Esophagus, atresia of.....1342
atresia of, congenital, with tracheal anastomoses 1296
atresia of, congenital, with tracheo-esophageal fistula 404
atresia of, 3 cases..... 609
congenital diverticulum of..... 607
diffuse dilatation of, due to trauma during birth 159
effect of certain drugs on..... 595
foreign bodies in, roentgen diagnosis of.. 429
foreign bodies in upper air passages and 639
septic ulceration of, in a new-born infant 898
Exsiccosis, experimental, residual oxygen in 600
Eye, congenital absence of abduction..... 186

Eye—Continued PAGE

growth of, and development of myopia; study in changes of refraction during school period 188
ophthalmic school clinics..... 878
pupillary membrane and congenital ectopia lentis 878
Eyelids, vacciniform blepharitis due to an atypical herpes virus; case..... 638

Faber, H. K.: A weight range table for children from 5 to 15 years of age....*758
Face, paralysis: See Paralysis, facial
Fasting, chemical changes in blood during, and subsequent refeeding; inorganic components 1285
Fats and chocolate, biologic effects of, irradiated by ultraviolet rays..... 606
effect of irradiated substances, especially fats 1295
food value of.....1291
metabolism in peripheral tissue.....1294
process of adipolysis in course of loss of weight in infancy..... 604
subcutaneous, frequency of atrophy of, following injection of insulin.....*715
tissue, influence of nutrition on composition of 1294
Favus of lid and scalp in infant 16 days old 200
Feces brilliant-green enrichment methods in examination for organisms of enteric group 147
calcium-soap 1293
diagnostic value of determinations of organic acids and ammonia in feces of infants 1289
Feeblemindedness, early diagnosis of mental defect 1313
in young, white blood cell picture and capillaries of nail groove in.....1314
psychologic tests in various types of congenital mental deficiency.....1321
Femur, epiphysiolysis of femoral head at birth 1109
Fetus, human, digestive enzymes of..... 399
inflammation of placenta and fetal sepsis 847
in utero, effects of x-ray and radium upon 404
Fever, obscure, in children.....1308
periodic pathothermy 1099
Fibula, gummatous ostitis of upper end....1304
Fingers, ainhum-like constriction of, with hyperkeratosis palmaris et plantaris...1321
Fischer, A. E.: Frequency of atrophy of subcutaneous fat following injection of insulin *715
Treatment of juvenile diabetes.....*323
Fistula, arteriovenous, congenital, in tympanum 1105
aural, congenital 1339
of bladder with osteomyelitis.....1334
tracheo-esophageal, congenital atresia of esophagus with 404
Flatfoot: See Foot, flat
Flatteau's Sign: See Meningitis, infantile
Fluoresceine-Sodium, uranin test as aid to diagnosis of meningitis.....185, 1318
Folin's method of determining amino-acids in blood.....1286
Foot, deformities and degeneration of..... 425
flat, juvenile.....*1273
Foreign Bodies, changes caused by, simulating tuberculosis.....1091
in chest..... 893
in esophagus, roentgen diagnosis of..... 429
in nose, method of removing..... 428
in upper air passages and esophagus..... 639
pipe stem in left main stem bronchus....1105
roentgen observations on safety pins as.. 427
tracheal, simulating diphtheria..... *95
Fossa Pharyngea: See under Pharynx
Fractures, temporary osteosynthesis in children 1108
Frisch, I. A.: "Blood cultures after tonsillectomy" *1282
Fruit juices, effect of acid and, on behavior of excised intestines..... 601
poisoning, etiology, symptomatology, pathology and treatment..... 154
Functional disorders in childhood..... 676

PAGE

Games of defective children.....1110
Gangrene of forearm in new-born infant... 162
spontaneous, of both legs.....1347
Garner, V. C.: Multiple disseminated lupus vulgaris; sequel to the exanthemas...*1028
correction to above article.....1235
Gastro-intestinal diseases in nurslings.....1291
Gaucher's Disease: See under Anemia
Geroderma, genitodystrophicum in children. 420
Glandular Fever, acute, of Pfeiffer..... 612
Glaucoma, pseudoglaucoma..... 186
Glomerulonephritis: See Nephritis, Glomerular
Glucose: See Dextrose
Glycerin in epidemic impetigo neonatorum..1075
Goiter, anatomy of..... 866
blood supply of thyroid gland with special reference to vascular system of cretin goiter 843
malignant, pathologic anatomy of, and of thyroid gland of cretins..... 867
Goldbloom, A.: Icterus neonatorum..... *57
Goldman, I. B.: Direct laryngoscopy as method for cultural studies of pulmonary secretions..... *47
Gonorrheal arthritis in new-born.....1077
Gordon, M. B.: Moro embrace reflex in infancy; its incidence and significance.. *26
Gottlieb, R.: Icterus neonatorum..... *57
Grass grown in dark, antirachitic principle in 1291
Graves, G. W.: Unusual association of postinfantile rickets with osteomalacia.... *84
Grayzel, H. G.: Influence of ultraviolet rays, yeast and irradiated yeast on sugar tolerance of diabetic children...*513
Management of ambulatory diabetic child.*275
Greenwald, H. M.: Congenital essential thrombocytopenia *1245
Diaphragmatic hernia in infancy and in childhood *361
Grip: See Influenza
Growth, body, of child, effect of ultraviolet light on.....155, 431
in length and weight of feebleminded children 638
influence of nucleic acids of various origin on 1288
of infants from standpoint of physical measurements and of nitrogen metabolism; creatine.....*499, *507

Hair, hereditary ectodermal dystrophy.....1321
Halpern, L. J.: Typhoid fever in children; study of 67 cases..... *10
Hamilton, B.: Acidosis of acute diarrhea in infancy.....*314
Handley, H. E.: Chronic tetany and chronic mycelial stomatitis in child aged 4½ years *328
Hassall's Corpuscles: See Thymus
Headache in children, treatment..... 171
Heart, abnormal ridge in left ventricle..... 595
block, congenital.....1094
block, congenital; report of case with incomplete heterotaxy; electrocardiogram in detrocardia.....*112
clinical method of estimating cardiac efficiency in children and work of a rheumatic supervisory center..... 858
congenital atresia of aortic ring..... 156
dextrocardia, congenital..... 608
dextrocardia, congenital, with transposition of viscera.....1078
Disease: See also Arrhythmia; Mitral Valve, stenosis; etc.
disease, digitalis studies on children with.*699
disease, the child with potential..... 628
disease, urobilinuria in.....1094
displacement in pneumonia.....1309
ectopia cordis.....*563
ectopia cordis pectoralis.....1296
ectopia cordis pectoralis; electrocardiographic study.....1297
extensive, diffuse, hydropic degeneration of myocardium associated with a diphtheritic infection..... 612
growth of human vascular system; a quantitative study with some functional implications 648

Heart—Continued PAGE
lesion, congenital, porencephaly with myoclonus and..... 874
mitral stenosis, x-ray diagnosis of..... 857
rate, paroxysmal tachycardia.....1099
rate, tachycardia with syncope occurring in a child.....*551
rheumatic carditis with complete heart block 632
rupture of, from abscess..... 858
valvular lesions, recovery from.....1094
Heine-Medin's Disease: See Poliomyelitis
Hejinian, L. M.: Growth of infants from standpoint of physical measurements and of nitrogen metabolism; creatine.....*499
Growth in infants from standpoint of physical measurements and nitrogen metabolism; uric acid.....*507
Heliotherapy, effect of outdoor schools and sunbathing on Chvostek sign in infants 180
Helmholz, H. F.: Spontaneous bacilluria and pyelitis in rabbit; relation to mode of infection in disease of urinary tract.*968
Helwig, F. C.: Imperforate anus with exit through prostatic urethra; report of 3 cases *559
Hemangioma of meninges involving visual cortex 1313
Hematoma, intracranial, in new-born.....1078
Hematoporphyria congenita..... 175
Hemiplegia after diphtheria..... 408
infantile, and mental retardation in hereditary syphilis.....1089
Hemoglobin: See also Blood; Erythrocytes
resistance as index of age of nurslings and prematurely born..... 150
Hemoglobinuria, paroxysmal; a review..... 629
Hemophilia, rate of disintegration of blood platelets in normal children in thrombopenia and..... 600
Hemorrhage, cerebral birth, in premature and immature infants..... 609
examination of blood in newborn and treatment of.....1075
of new born..... 850
prenatal prevention of potential disease of new-born 849
Hemotherapy and exudative diathesis..... 631
Heredity, unusual case of familial disease.. 179
Hernia, diaphragmatic, in infancy and in childhood *361
inguinal, and undescended testicle in single ovum twins.....1078
inguinal, operation for..... 645
internal, in infant aged 3 weeks..... 885
strangulated, in early infancy.....645, 1329
Herpes, herpetic infection, with especial reference to involvement of nervous system 640
virus, vacciniform blepharitis due to an atypical 638
zoster 897, 1322
zoster and varicella.....428, 1299
zoster and varicella; statistical observations 1299
Heuser, G. F.: Rickets in chicks; effect of heat and exposure to air on stability of vitamin D.....*481
Hexamethylamine: See Methenamine
Hip, congenital dislocation of, diagnosis and treatment of.....1114
dislocation, sudden, in paralyzed child on non-paralyzed side.....1326
Hirschsprung's Syndrome: See Colon
Hodgkin's Disease: See Lymphogranuloma
Hoffman, S. J.: Gaucher's disease; review of literature and report of case diagnosed from section of an inguinal lymph gland *775
Holt, L. Emmett, unveiling of memorial tablet to.....1112
Howland, John, bas-relief of, unveiled.....1281
Hucker, A. M.: Organisms associated with commercially prepared infant foods....*310
Hucker, G. J.: Organisms associated with commercially prepared infant food.....*310
Huenekens, E. J.: Mental hygiene from a pediatric standpoint; a study of 400 cases in a preschool clinic.....*824
Hulvey, C. N.: Relations of weight, height and age in negro children.....*1222
Hurt, A. S., Jr.: Anomalies of urinary tract in infants *1202

PAGE
Hydrocephalus, pathogenesis of..... 184
treatment of..... 877
Hydronephrosis, congenital, and pseudohermaphrodism 1338
Hygroma: See Bursa
Hyperkeratosis palmaris et plantaris with ainhum-like constriction of fingers.....1321
Hypertelorism, unilateral..... 416
Hypophysis: See Pituitary Body
Hypotrophy, syphilitic.....1088

Icterus: See Jaundice
Ichthyosis congenita and ichthyosis vulgaris; heredity 642
Idiocy, amaurotic family, (Tay-Sach's disease) notes on pedigree of..... 873
mongolian, diabetes in..... 874
mongolian, etiology of mongolism..... 178
mongolism, with enlarged liver and jaundice 874
Immunity, function of spleen in.....1075
natural acquirement of..... 598
Immunization from the public health standpoint 408
Impetigo bullosa; case report..... 642
neonatorum, epidemic glycerin in.....1075
Infant Feeding..... 605
breast-feeding and living conditions of mothers 156
curd and buffer in..... 602
development of premature infants in feeding stations..... 404
first application of new-born infant to breast 611
general indications for choice of formulas for 401
organisms associated with commercially prepared infant foods.....*310
physiology of suckling and proper shape of nipple for bottle.....1072
routine use of vitamin B factor in..... 602
Infant Mortality: See also Vital Statistics
and birth rate in Bavaria..... 890
in Mecklenburg before and after the war. 431
in Oregon, decrease in..... 888
mortality list of infants during 330 years.1109
statistical report for 1928.....1323
stillbirths and neonatal deaths in 1,960 consecutive private cases..... 450
summary of, in registration area: 1928.. 888
Infant, New-born: See New-Born Infants
Infant Welfare, international congress for protection of infancy..... 647
protection of infants in Montevideo.....1328
Infanticide by mother during birth..... 200
Infantilism, celiac: See Celiac Disease
Infections, congenital..... 855
institutional, and prophylactic "cold" vaccines 1345
occurrence of primary purulent meningitis with acute infections.....1317
some types of, in newly born..... 609
Infectious Diseases: See Communicable Diseases
Influenza, grip associated with a morbilliform rash and a septic termination in new-born 405
Injections, intragluteal, fate of..... 622
Insulin, absorption of, from mucous membranes of mouth..... 175
frequency of atrophy of subcutaneous fat following injection of.....*715
local fat dystrophy after injections of... 420
Intelligence, relation between mental and physical status of children in 2 counties 888
Intestines, association of bowel disease with vitamin C deficiency..... 155
epidemic of intestinal infections with bacteremia and toxemia in infants at Warsaw 1300
excised, effect of acids and fruit juices on behavior of..... 601
intussusception and volvulus, rheumatoid purpura complicated by..... 632
occlusion, of complex origin..... 645
paralytic ileus and high intestinal obstruction; their rational management.. 884
passage of living bacteria through wall of 143
pneumatosis *818

PAGE

Intussusception, acute, in infants; 8 cases. 885
and volvulus, rheumatoid purpura complicated by..... 632
in infant aged 6 months..... 645
non-operative treatment of..... 429
with rupture and peritonitis.....1334
Iowa Child Welfare Research Station, State University of Iowa.....*541
Iron in blood and other liquids, accurate micro-determination of.....1074
Iso-Agglutinins: See Agglutinins
Isohemagglutination: See Blood, groups
Ivimey, M.: Bone dystrophy; with characteristics of leontiasis ossea, osteitis deformans and osteitis fibrosa cystica in child. Suggestion as to influence of central nervous system.....*348

Jampolis, M.: Ureteral neuromuscular dystrophy in relation to infections of kidneys *1006
Jaundice and enlarged liver, mongolism with 874
grave familial, of newly born..... 405
icterus neonatorum..... *57
icterus neonatorum as consequence of isoagglutination 1077
relationship of weight and, to blood values in new-born infant.....*518
simple icterus of new-born..... 450
Jejunum, duplex.....1079
Joints, hereditary symmetrical hypoplasia of 161
Josephs, H., Spontaneous hypoglycemia in childhood *746

Kahn Precipitation Test; comparative clinical study with Noguchi-Wassermann reaction *1206
for syphilis in childhood.....1115
Kajdi, L.: Acidosis of acute diarrhea in infancy *314
Kala-Azar: See Leishmaniasis
Kaucher, M.: Metabolism of undernourished children; effect of high and low protein diets on excretion of creatine, creatinine and ammonia.....*468
Metabolism of undernourished children; study of basal metabolism, caloric balance and protein metabolism during a period of gain in weight.....*476
Keratoconus of endocrine origin, pluriglandular opotherapy in.....1104
Kern, R.: Metabolism of undernourished children; study of basal metabolism, caloric balance and protein metabolism during a period of gain in weight.....*476
Kidney, abscess, paranephric..... 887
abscess, perirenal, in infancy..... 419
adenoma of..... 857
anatomical changes in..... 595
calculi during first years.....1101
congenital one-sided defect of, with presentation of 3 new cases of agenesia and 1 of aplasia..... 156
congenital polycystic, arterial supply of.. 145
horseshoe *799
infections, ureteral neuromuscular dystrophy in relation to.....*1006
juvenile nephrosis..... 862
lipoid, bacteremia and skin manifestations in nephrosis.....*762
metabolic studies on nephrosis, with particular reference to nitrogen balance.. 650
nephrosis and nephritis.....1100
Kiyasu, K.: Urticaria pigmentosa; report of nodular case and review of literature.*1020
Klipper-Feil Syndrome: See Spine
Knee, congenital backward dislocation of..*107
Kohn, J. L.: Bacteremia and skin manifestations in lipoid nephrosis.....*762
Successive roentgenograms of chest of children during measles.....*258, 1118
Koiransky, H.: Successive roentgenograms of chest of children during measles.*258, 1118
Kotzen, H. F.: Progeria; review of literature and case report.....*993

PAGE

Kreidel, K. V.: Kahn precipitation test in infancy and early childhood; comparative clinical study with Noguchi-Wassermann reaction.....*1206
Kretchmer, H. L.: Congenital valves of posterior urethra.....*804

Lange reaction in diagnosis of congenital syphilis 1304
Laryngitis, acute streptococcic, with edema; tracheotomy cure..... 882
Laryngoscopy, direct, as method for cultural studies of pulmonary secretions..... *47
Larynx, carcinoma of, in young..... 428
occlusion by ascaris lumbricoides, sudden death due to..... 192
papillomatosis of trachea and..... 639
postdiphtheritic stenosis of cicatrices of.. 164
smears from..... 414
Lederer, M.: Splenomegaly; portal phlebosclerosis *1231
Lee, P.: Sickle cell anemia in a Greek family *103
Leishmaniasis, infantile kala-azar..... 174
infantile kala-azar; case cured at Alexandria by new method..... 857
Lens, crystalline, oxidation mechanism of.. 191
Leontiasis ossea, bone dystrophy with characteristics of, in child.....*348
Lestocquoy, C.: Diagnosis of tuberculosis of tracheobronchial glands.....*1125
Leukemia, borderline cases of pernicious and aplastic anemias and..... 632
in infancy; 3 cases..... 860
Leukocytes and capillaries of nail groove in young mental defectives.....1314
in measles.....620, 855
response to throat infection..... 881
Leukopenia with infectious mononucleosis.. 658
Levulose test in rheumatic and choreic children 1081
Lichen planus, generalized, cured by radiation of axilla (peri-arterial sympathetic radiotherapy) 884
Light, influence of environment on growth and nutrition of normal rabbits, with especial reference to action of neon light 146
effect of, on blood and tissue cells..... 146
influence of environment on normal rabbits; calcium and inorganic phosphorus in blood of rabbits..... 597
Lipodystrophia progressiva.....1096
Lipoids, influence of, on action of diphtheria toxin in animal experiments..... 411
lipoid nephrosis, bacteremia and skin manifestations in.....*762
Lipoma, congenital, of femoral vein..... 159
Little's Disease: See Paralysis, spastic
Liver, acute red atrophy in child..... 861
cirrhotic change in, and carbohydrate metabolism disturbances.....1092
enlarged, and jaundice, in mongolism.... 874
hepatic cirrhosis with ascites and splenomegaly; complete relief from ascites under antisyphilitic treatment..... 624
hereditary syphilis of, anatomic forms of.1088
solitary nonparasitic cysts of.....*241
therapy of anemias.....632, 1097
Longevity, effect of nucleic acids of various origin on.....1288
Lues: See under Syphilis
Lung, abscess, experimental production of 669, *683
cyst formation in.....1311
diagnostic value of D'Espine's sign in adults 431
direct laryngoscopy as method for cultural studies of pulmonary secretions..... *47
diseases in childhood, diagnostic errors in 861
fibrosis in children, discussion of..... 623
interpretation of roentgenograms of chest based on necropsy observations..... 208
pathologic changes in large interlobar fissures in children..... *75
postnatal development of.....1072
Tuberculosis: See Tuberculosis, pulmonary
Lupus vulgaris, multiple disseminated, following the exanthemas.....*1028
correction to above article.....1255

PAGE

Lymph Nodes, abdominal, tuberculosis of..1088
diagnosis of tuberculosis of tracheobronchial glands, with anatomic control.... 648
inguinal, Gaucher's disease, review of literature and report of case diagnosed from section of.....*775
mediastinal lymphadenitis.....1094
primary generalized round cell sarcoma of 860
roentgen ray treatment of lesions of lymphoid tissue..... 430
tuberculous cervical adenitis.....1302
tuberculosis of tracheobronchial glands, diagnosis of.....*1125
Lymphadenitis: See under Lymph Nodes
Lymphatism, reported hypoplasia of suprarenal gland in so-called status thymicolymphaticus 1097
Lymphogranuloma, lymphoblastoma (Hodgkin's disease) of orbit.....1104
rationale of radiotherapy in Hodgkin's disease and lymphosarcoma..... 415
Lymphosarcoma: See Sarcoma
Lyttle, J. D.: Prognosis of acute nephritis.*1052

McKenzie, M. R.: Influence of ultraviolet rays, yeast and irradiated yeast on sugar tolerance of diabetic children...*513
McKitterick, J. C.: Pneumonic anthrax in a child; report of a case.....*1252
MacLean, A. B.: Carbohydrate tolerance in infants and in young children with celiac disease.....*16
McQuarrie, I.: Epilepsy in children; relationship of water balance to the occurrence of seizures.....*451
Epilepsy in children; reply to Dr. Peterman *1068
Magnesium, calcium and potassium contents of spinal fluid.....1320
Makler, M. I.: Gaucher's disease; review of literature and report of case diagnosed from section of an inguinal lymph gland.....*775
Malacoplakia of bladder, etiology of..... 865
Malaria, prolonged pneumococcus septicemia suggestive of..... 854
report of 3 cases.....1340
treatment of congenital syphilis....414, 1305
treatment of gonorrheal vulvo-vaginitis.. 616
treatment of, with plasmochin..... 410
Malformations: See Abnormalities
Malnutrition: See under Nutrition
Mastoiditis, acute, and acute middle ear suppurations, bacteriology and clinical course, and complications..... 197
acute, in an infant aged 8 weeks.....*567
acute, resorption of bone in..... 640
Maternity and child welfare with special reference to legislation..... 202
Maxillary Sinus: See Antrum
Measles and the common cold..... 407
behavior of white blood corpuscles in.... 620
disseminated tuberculosis verrucosa cutis following 1323
followed immediately by scarlet fever....1330
hemorrhagic preexanthems; a new sign in 411
illness of infection in..... 408
pooled adult blood serum as prophylactic measure in 219
prophylaxis of 1085
prophylaxis with reactivated serum of adults 1085
sensitiveness of children with, to cutaneous injections of epinephrine.... 616
streptococcus toxin and antitoxin, clinical results with 612
successive roentgenograms of chest of children during *258, 1118
symptomatic purpura hemorrhagica after rubella, ... 409
white cells in 855
whooping cough and chicken pox, completeness of reporting of, at different ages 614
Measurements of children: See Anthropometry
Mediastinum, lymphadenitis of.....1094
Meeker, D.: Acidosis of acute diarrhea in infancy *314

PAGE

Megacolon: See Colon, Dilatation
Megalosyndactilitis: See Abnormalities
Melena neonatorum, blood in 1297
Memory, infantile, reliability of..... 871
Meninges, hemangioma of, involving visual cortex 1313
Meningiomas of cribriform plate, ocular trouble in 874
Meningitis, epidemic cerebrospinal, with specific antimeningococcic serum 166
in children caused by Koch-Weeks' bacilli 184
in new-born infants 406
infantile, Flatteau's sign in..... 876
influenzal, in infants 425
lactic acid of spinal fluid as prognostic and therapeutic guide in 211
meningeal permeability and diagnosis of, ... 183
meningococcic, and measures for its control 853
meningococcus, clinical study of 207
meningococcus, determination of value of serum in treatment of 210, *730
meningococcus, treatment by combined cisternal and lumbar medication..... 423
Neisseria subflava (Bergey), in infant.. 176
primary purulent, following acute infections 1317
symptoms of 1317
tuberculous, diagnostic value of cisternal puncture and ventriculography in.....1089
tuberculous, lactic acid content and p_H of spinal fluid in 414
tuberculous, pathogenesis of 1090
uranin in diagnosis of 185, 1318
Meningococci infection in infants 616
septicemia, fulminating, without meningitis 1299
Meningo-encephalomyelitis, acute, of childhood 635
Mental defectives: See under Feeblemindedness
Mental Diseases, congenital syphilis and juvenile psychopathies 1089
significance of encephalitis in origin of mental defects 1103
value of child training in prevention of mental disorders 871
Mental Hygiene from a pediatric standpoint *824
Mental Tests, psychologic tests in various types of congenital mental deficiency..1321
Mercurochrome, study of action of horse serum and, on pneumococcus infections in mice 596
Merritt, K.: Tuberculosis in infants under 1 year of age; study of autopsy and clinical observations on infants with tuberculous lesions *526
Meta: See Acetaldehyde
Metabolism, basal and protein, and caloric balance, study of, during a period of gain in weight *476
basal, in children and adolescents 403
basal, in children of abnormal body weight; in underweight children.....*299
of undernourished children; effect of high and low protein diets on excretion of creatine, cretatinine and ammonia *468
protein and fat, influence of administration of thyroid on 599
water, in infantile eczema 642
Methenamine, urinary disturbances in infant caused by hexamethylenamine.... 419
Mettel, H. B.: Antidiuretic effect of solution of pituitary dropped intranasally in case of diabetes insipidus.....*342
Microcephaly in twins 1296
radium therapy of uterine cancer during pregnancy followed by microcephalus..1102
Microphthalmos, congenital bilateral, accompanied by other malformations..... 185
Migraine in early infancy 636
Milk, acidified, attempt to substitute for buttermilk in feeding of debilitated and hypothreptic newly born infants..... 604
Brucella abortus in, as source of agglutinins in human sera 1073

Milk—Continued PAGE

Brucella abortus in, thermal death point of 597
elimination of nicotine in 152
epidemiology of an outbreak of illness due to 405
feeding of children 152
human, antirachitic factor content of; report on efforts to increase it 442
human, drying up of breasts 430
human experimental study of excretion of morphine in 606
human, intoxication, infantile beriberi with symptoms of 156
human, iso-agglutinins in 1292
human, registry of 140
human, studies on composition of 152
irradiated, or irradiated ergosterol?.... 606
laboratory control of, under new state rule 603
nature of proteins surrounding fat globules in 1285
phosphorus compounds of; presence of adenine nucleotide in milk 1287
sulphur in human and cow's, ... 605
Weissenberg's citric acid whole, as a prophylactic against rickets 402
Miller, J.: Tracheal foreign body simulating diphtheria *95
Mitchell, J. M.: Relationship of jaundice and weight to blood values in newborn infant *518
Mitral Valve, stenosis, x-ray diagnosis of.. 857
Moersch, H. J.: Cardiospasm in infancy and in childhood *294
Money, vitality of vibrio cholera on Chinese paper money 1088
Mongolism: See Idiocy, Mongolian
Mononucleosis, infectious, with leukopenia.. 658
Monsters (Zrudy) 405
Moore, R. A.: Intestinal pneumatosis *818
Morphine, experimental study of excretion of, in breast milk 606
Mortality: See Death Rate
Movements, methods for mass examination of motor functions of children and minors 636
muscular, in premature and young infants 1072
Muller, H.: Basal metabolism in children of abnormal body weight; in underweight children *299
Mumps: See Parotitis
Muscles, alterations in, observed in syphilitic osteopathies of infancy 413
neuro-muscular coordination in children, some disorders in 634
Myatonia, amyotonia congenita and spinal muscular atrophy of Werdrig-Hoffmann in 1 family 185
familial amyotonia congenita..... 632
Mycosis, "fruit poisoning," etiology, symptomatology, pathology and treatment... 154
of palpebral conjunctiva 189
Myers, C.: Otitis media in infancy; diagnosis by means of cultures taken from middle ear; description of method *49
Myocarditis, interstitial 859
Myocardium, alterations in, in infectious diseases 1310
Myopia, growth of eye and development of; study in changes of refraction during school period 188
race, sex and environment in development of 187
school, does it exist? 880
school or occupational, does it depend on close work or is some other factor responsible for it 881
Myotonia congenita 1319
Myxedema and maldevelopment following cerebral injury at birth.....1334
congenital, Hirschsprung's syndrome as first sign of 861
infantile, thyroxin in 421
protein content of spinal fluid in 145
thyroid gland preparations for 1312

PAGE

Nail, capillaries of nail groove and white blood cell picture in young mental defectives 1314
finger and toe, of nurslings 884
hereditary ectodermal dystrophy 1321
pachyonychia congenita 1107
Narcolepsy: See Sleep
Narcotic substances, use of, in children... 202
Negro children, relation of weight, height and age in *1222
Nelson, M. V.: Dietary control of dental caries *721
Neoarsphenamine: See Arsphenamine
Nephritis and nephrosis 1100
acute, in children, intermediated prognosis of 651
acute, prognosis of *1052
chronic 1340
empyema, pneumonia and pneumococcal peritonitis with complete recovery....1309
glomerular, experimental acute, ...1343
varicella complicated by; report of case associated with streptococcal infection of tonsils 852
Nephrosis: See under Kidneys
Nerve, optic, unilateral atrophy of, in congenital syphilis 1304
Nervous System, central, birth traumas of.. 405
central, influence of, on bone dystrophy with characteristics of leontiasis ossea, osteitis deformans and osteitis fibrosa cystica in child *348
herpetic infection, with especial reference to involvement of 640
in rats, fed on diets deficient in vitamins B and B_2 845
sympathetic, relation between congenital cataract and changes in neurovegetative apparatus 190
sympathetic, and specific dynamic action. 150
vegetative, effect of infections on..... 877
Neuraxitis Epidemica: See Encephalitis, epidemic
Neurosyphilis in the young, treatment 170
Nevi of bulbar conjunctiva 186
New-Born Infants: See also under names of diseases, i. e., Melena neonatorum; Sclerema neonatorum; etc
cardiospasm in, report of 3 cases *1183
effect on, of irradiating mother during pregnancy 611
examination of blood in, and treatment of hemorrhage 1075
intracranial hemorrhage in, as demonstrated by roentgen rays *1196
lines in face of 611
living blood cells of, ...1297
studies in height, weight and sitting height of 610
Nicotine, elimination of, in milk 152
Niemann's Disease, lipoid cell splenohepatomegaly (Niemann-Pick) 862
Nipple, proper shape of, for bottle and physiology of suckling at breast.....1072
Nitrogen, balance, metabolic studies on nephrosis with particular reference to 650
metabolism, growth of infants from standpoint of physical measurements and of *499, *507
Norris, L. C.: Rickets in chicks; effect of heat and exposure to air on stability of vitamine D *481
Nose, congenital defects of vomer and development of deformities of septum.. 194
foreign bodies in, method of removing.. 428
obstruction in childhood (pseudovegetations) diastolization in 193
otitis after packing of nasopharynx and 194
Nucleins, influence of nucleic acids of various origin on growth and longevity of white mouse 1288
Nursery school, medical aspects of.....1337
Nutrition and tonsils 1344
digestive and nutritional disturbances in the nursling 1291
disturbances in infancy..... 403
malnutrition in children 1285

Nutrition—Continued PAGE
metabolism of undernourished children; study of basal metabolism, caloric balance and protein metabolism during a period of gain in weight....*476
nutritional edema 1291
of twins from one ovum 606
requirements of sick children 1333
the undernourished child; analysis of results of convalescent care 648

OBITUARIES:
Francioni, Carlo 398
Guinon, Louis 398
Heim, Paul 1281
Outhouse, Alice 398
Rachford, Benjamin Knox. 1857-1929.... 141
Oculogyric Crises: See under Encephalitis, epidemic
O'Donnell, W. S.: Otitis media in infancy; its diagnosis by means of cultures taken from middle ear; description of method *49
Ophthalmia nodosa (caterpillar hair ophthalmitis) 880
Optochin (Ethyl Hydrocuprein): See under Empyema
Orange juice, vitamins of; vitamins of commercially concentrated orange juice....1286
Orbit, lymphoblastoma (Hodgkin's disease) of 1104
Os vesalianum pedis 1072
Osgood-Schlatter's Disease: See under Tibia
Osteitis, bone dystrophy with characteristics of leontiasis ossea, osteitis deformans and osteitis fibrosa cystica.....*348
gummatous, of upper end of fibula.....1304
Osteochondritis luetica; comparison with analogous zones in rickets and infantile scurvy 622
Osteomalacia, unusual association of post-infantile rickets with *84
Osteomyelitis 645, 1341
acute, and complications.... 644
acute, followed by purulent pericarditis..1095
acute, with complete sequestration of long bones 671
bilateral, of tibia 885
infectious; differential diagnosis 1107
with fistula of bladder 1334
Osteosynthesis: See under Fractures
Otitis Media after packing of nose and nasopharynx 194
in infancy, diagnosis by means of cultures taken from middle ear; description of method *49
mucosus, ... 198
Owens, R. W.: Bilateral congenital facial paralysis; review of literature on congenital facial paralysis and a classification *1256
Oxygen, residual, in experimental exsiccosis 600
subcutaneous injections of, to improve appetite and nutrition in infants..... 604
Oxyuris infestation of man, 2 rare observations 175

Pachyonychia congenita 1107
Pain, epigastralgic syndrome of vertebral and intravertebral origin.... 633
Palms, hyperkeratosis palmaris et plantaris with ainhum-like constriction of fingers 1321
Palsy: See Paralysis
Papillomatosis of larynx and trachea..... 639
Paralysis, cerebral diplegias, rôle of arterial occlusion and anoxemia in cause of.. 423
facial, and congenital defects of radius and ear.... 161
facial, congenital; review of literature and a classification....*1256
infantile, acute epidemic (Heine-Medin) in East Prussia.... 619
infantile cerebral palsies.... 180
infantile cerebral palsy illustrating effect of active movement on spasm.....1103
infantile, clinic, in Tennessee..... 837
of respiratory centers, stimulant drugs in 427

Paralysis—Continued PAGE
partial, of lower extremities, in children, accompanied by backward mental development 870
spastic, Little's disease in sisters and brothers and in twins.... 182
spastic, sympathetic ramisection in..... 644
Paraplegia with Pott's disease..... 887
Parasites, duodenal, in infant..... 621
Parotitis, immunization experiments in epidemic of..... 620
Peanut Flour, value of, in infant feeding.. 605
Pearson, G. J.: Pneumonic anthrax in a child; report of a case.....*1252
Pediatrics, preventive, and the American Pediatric Society..... *1
problems of pediatricians as the psychologist sees them..... 676
Second International Pediatric Congress in Stockholm 837
Pellagra as observed in China..... 607
Pemphigus, congenital, apparently not of syphilitic origin..... 884
hereditary 429
neonatorum, epidemic; method of treatment 609
neonatorum in hospitals..... 437
Pericarditis and septicemias due to streptococcus 859
chronic adhesive 662
precordial thoracectomy for adherent pericardium 886, 887
purulent, following acute osteomyelitis..1095
rheumatic, and pancarditis of Bouillaud's disease 859
Pericardium, puncture via the epigastrium. 889
Volhard's stasis following concretion cordis 1310
Peritonitis following intussusception with rupture 1334
pneumococcal, nephritis, empyema and pneumonia with complete recovery....1309
pneumoccic; report of case..... 633
pneumococcus, in infants..... 1309
Perspiration: See Sweat and Sweat Glands
Pertussis: See Whooping Cough
Peterman, M. G.: Epilepsy in children...*1068
Horseshoe kidney *799
Pharmacology, recent advances in..... 598
Pharynx, frequency and distribution of fossa pharyngea, in human crania.... 143
pharyngeal hypophysis and adenoids.... 193
Phenocoll hydrochloride in whooping cough and congenital stridor of new-born...1082
Phenylethylhydantoin: See Barbital and Barbital Derivatives
Phlebosclerosis, portal.....*1231
Phosphorus, bone changes in continuous ingestion of 1344
compounds of milk; presence of adenine nucleotide in milk.....1287
metabolism, disturbances of, in rickets; excretion of endogenous phosphate....1295
metabolism in rickets; phosphatemic curve after parenteral introduction of phosphorus 1295
phosphatemic curve of normal and rachitic organisms 1295
Photomicrographs, reproduction of..... 840
Pierson, L. E.: Congenital valves of posterior urethra *804
Pituitary Body, glandular elements in posterior lobe of human hypophysis..... 595
is secretion of, concerned in inheritance of body-size? 143
some properties of separated active principles of pituitary (posterior lobe)... 147
suprapituitary tumor; 2 cases..... 416
Pituitary Extract, antidiuretic effect of solution of, dropped intranasally in case of diabetes insipidus.....*342
Placenta, inflammations of, and fetal sepsis. 847
Plasmochin: See Malaria
Pleonosteosis: See Bones, growth
Pleurisy, acute serofibrinous, pathogenesis of *829
corticopleuritis 1102
hemorrhagic, in child 631
Pneumatosis, intestinal *818

PAGE

Pneumococcemia and pulmonary tuberculosis 1090
in child aged 4 years 615
with multiple manifestations 615
Pneumococci infection, treatment with biliary salts 416
infections in mice, study of action of mercurochrome, of horse serum and of mixtures thereof on 596
septicemia, prolonged, suggestive of malaria 854
types of pneumococcus in empyema of childhood 175
Pneumonia, congenital nonspecific, and pneumonia from aspiration in the birth passage 848
croupous, and bronchopneumonia 628
croupous, in infants and children, white blood cell picture of 869
displacement of heart in 1309
empyema, nephritis and pneumococcal peritonitis with complete recovery 1309
encephalitis complicating 1103
in childhood 1073, 1308
in new-born infant 894
lobar, bronchoscopic findings in 856
lobar, frequency of 1080
pneumonic syndrome in nurslings 1081
studies 1096
Pneumothorax, simulation of, by thoracic rickets 402
spontaneous, in children under 2 years 631
Poliomyelitis, convalescent serum in 594
convalescent serum in, résumé of results of therapy with 163
experimental, blood sedimentation test in 841
experimental, relation of streptococci to spinal fluid in 596
Heine-Medin's disease in light of observations at autopsy; prognosis of 878
in Syracuse 614
incubation period of 852
serum therapy in 164
survival of virus of, for 8 years in glycerol 842
Polycythemia, cyanosis of, in child aged 6 years 174
Polydipsia: See Thirst; Diabetes Insipidus
Polyneuritis, postdiphtheritic 635
Polyserositis 657
Porencephaly with myoclonus and congenital heart lesion 874
Potassium, calcium and magnesium contents of spinal fluid 1320
Pott's Disease: See Spine, tuberculosis
Pregnancy and syphilis 1301
effect on new-born infant of irradiating mother during pregnancy 611
epidemic encephalitis in labor, puerperium and 1075
management of syphilitic pregnant women; its relation to prognosis for the child 625
radium therapy of uterine cancer during, followed by microcephalic idiocy 1102
roentgen or radium irradiation during, outcome in 625 cases 1324
Premature birth, carbohydrate metabolism of premature infants; blood sugar during fasting *912
continuous temperature records of premature infants *944
development of premature infant in feeding stations 404
muscular movement in premature 1072
problem of premature infant 157
Primogeniture, is it a factor in production of developmental anomalies? 607
Progeria, review of literature and case report *993
Protein cereals and "aleurons" 1290
of eggwhite 845
Prurigo—eczema—asthma complex 198, 199
Pseudoglaucoma: See Glaucoma
Pseudohermaphrodism and congenital hydronephrosis 1338
Pseudoparalysis of Parrot 1089, 1090
Psychology, value of child training in prevention of mental disorders 871

PAGE

Purpura hemorrhagica gangrenosa *1013
hemorrhagica, symptomatic, after rubella. 409
rheumatoid, complicated by intussusception and volvulus 632
Pyelitis, acute, so-called, in infants, pathology of 214, *227
in children, present status in 629
in infancy 860
spontaneous bacilluria and, in rabbit; relation to mode of infection in urinary tract disease 432, *968
Pyelonephritis, treatment of 1310
Pyemia, glanders-like, in breast-fed new-born child 851
Pylorus, congenital valves, pseudovalves and stenosis of duodenum and 846
obstruction, infantile; preliminary report of its allergic nature *741
pylorospasm 866
pylorospasm and cardiospasm in infant 1307
pylorospasm, roentgen treatment of 1311
stenosis, anatomic observations in 2 cases 885
stenosis, congenital; anatomic examination 3 months after pylorotomy 1327
stenosis, congenital hypertrophic 857
Pyonephrosis, bilateral, with calculi in an infant aged 13 months 633
Pyuria: See Urine

Quarantine regulations for diphtheria and scarlet fever, can they be less stringent than for measles and pertussis 1300
Quartz Lamp: See Ultraviolet Rays

Rachitis: See Rickets
Radium and x-rays, effects of, on fetus in utero 404
capsules in stomach; gastroscopic removal 1093
microcephalic idiocy following radium therapy of uterine cancer during pregnancy 1102
therapy during pregnancy, result of 1324
therapy, peri-arterial sympathetic, case of generalized lichen planus cured by 884
therapy, rationale of, in Hodgkin's disease and lymphosarcoma 415
use of, in diseased tonsils 639
Ragweed pollen and diphtheria bacilli, hypersensitiveness to 843
Ramisection, sympathetic, in spastic paralysis 644
Ray, H. H.: Fluorescent properties of ringworm of scalp as aid in diagnosis *339
Urticaria pigmentosa; report of nodular case and review of literature *1020
Reading ability, extraordinary deficiency in 1321
Reflexes, conditioned and unconditioned, method of investigation of 1318
conditioned, external inhibition of 1318
Moro embrace reflex, in infancy; its incidence and significance *26
Refraction, solid atropine and cocaine for 1104
Respiration, difficult, noisy and rapid, some causes of 1306
Respiratory Tract and middle ear infections 856
foreign bodies in esophagus and upper air passages 639
institutional infections and prophylactic "cold" vaccines 1345
stimulant drugs in paralysis of 427
susceptibility of Eskimos to common cold and study of their natural immunity to diphtheria, scarlet fever and bacterial filtrates 163
winter peak of respiratory diseases 1100
Reticulocytes in embryos and the new-born 851
Retina, embolus of central retinal artery 1321
vitamin A in retinal tissue compared with vitamin content of brain tissue 1074
Retinitis pigmentosa albescens 638
Rheumatic Fever 1340, 1345
allergic reactions with a toxin-producing strain of nonmethemoglobin-forming streptococcus isolated from 597

Rheumatic Fever—Continued PAGE
and tonsillitis 853
clinical method of estimating cardiac efficiency in children and work of a rheumatic supervisory center 858
effects of digitalis on electrocardiograms of children with chronic valvular heart disease and *699
Rheumatism 659
acute, in childhood, and its sequelae 614
carditis with complete heart block 632
effect of variation of diet in 1326
levulose test in rheumatic children 1081
problem of 1084
Rickets, acute and chronic 1291
and severe nutritional disturbances, effect on children's teeth 440
and tetany in rats 145
and thymus 672
antirachitic factor content of human milk; report on efforts to increase it ... 442
antirachitic principles in grass grown in dark 1291
disturbances in metabolism of phosphorus in; phosphatemic curve for rachitic and nonrachitic infants after parenteral introduction of phosphorus 1295
disturbances of phosphorus metabolism in; excretion of endogenous phosphate .. 1295
experimental 845
human, and rachitic spasmophilia, quartz lamp therapy in 602
in chicks, effect of heat and exposure to air on stability of vitamin D *481
method of assay of antirachitic vitamin D 1287
osteochondritis luetica, comparison with analogous zones in infantile scurvy and 622
phosphatemic curve of normal and rachitic organisms 1295
postinfantile, unusual association of, with osteomalacia *84
prevention by means of ultraviolet irradiation 601
properties of chondroid zone of rachitic bones 1290
rachitic caput natiforme, ... 402
relation of altitude of sun to its antirachitic effect 602
renal, ... 660
severe case of 401
so-called summer, critique of article by Schoenen 1292
thoracic, simulation of pneumothorax by 402
treatment and prevention with irradiated ergosterol 402
Weissenberg's citric acid whole milk as a prophylactic against 402
why it does not exist in Mexico ... 1288, 1289
Ringworm of scalp, fluorescent properties of, as aid in diagnosis *339
Roberts, M. H.: Intracranial hemorrhage in new-born infant as demonstrated by roentgen rays *1196
Roentgen Ray demonstration of intracranial hemorrhage in new-born infant *1196
diagnosis of foreign bodies in esophagus .. 429
diagnosis of lesions of upper urinary tract unassociated with calculus 627
effects of radium and, on fetus in utero 404
influence on white mice and their progeny 1073
observation on safety pins as foreign bodies 427
of chest, interpretation of, based on necropsies 208, *978, *1166
therapy during pregnancy, result of 1324
therapy, present status in pediatrics 1341
treatment of lesions of lymphoid tissue .. 430
Rosenberg, L.: Prognosis of acute nephritis *1052
Royster, L. T.: Relations of weight, height and age in negro children *1222
Rubella: See Measles
Rubin, M. I.: Blood cultures after tonsillectomy *726
Ruhräh, J.: J. Starsmare, 1664 ..... *1277
John Mayow, 1643—1679 ..... *590
Ruhräh, J.—Continued PAGE
Richard Wiseman, 1622-1676 ..... *137
Thomas Sydenham, 1624-1689 ..... *393
Walter Harris, 1647-1742 ..... *1064
Wolfgang Hoefer, 1614-1681 ..... *834
Sacrococcygeal tumor 174
Salt, new substitute for, and salt-free diet .. 404
Salyrgan, diuresis due to, and blood volume. 1098
Sanford, H. N.: Polymorphonuclear count in new-born; deflection by ultra-violet light *547
Polymorphonuclear count in new-born; preliminary report *271
Sarcoma, mesenteric lymphosarcoma 174
primary, generalized round cell, of lymph glands 860
rationale of radiotherapy in Hodgkin's disease and lymphosarcoma 415
Scarlatina: See Scarlet Fever
Scarlet Fever 410, 663
anaphylactic sensitization of guinea-pig to scarlatinal streptococcus filtrate 657
and allergy 613
and hemolytic streptococcus 411
and tonsillectomy 164
antiserum 409
blanching with placental serum *953
cardiovascular disturbances in 1087
control of *905
healthy carrier in 612
hemolytic streptococci in healthy persons and their importance in prevention of .. 616
histologic changes of thymus in children with 617
immediately following measles 1330
immunization 407, 1085
1895-1914 1079
opsonification test for rapid identification of streptococcus of 163
prevalence of, and susceptibility to, in North Carolina 1080
sensitiveness of children with measles and, to cutaneous injections of epinephrine .. 616
statistical study of diphtheria and 613
susceptibility of Eskimos to common cold and study of their natural immunity to 163
toxoid for immunization against 1332
value of Dick intracutaneous test for laryngorhinologic patients 196
Schauffler, G. C.: Comments on article "cervicovaginitis" by Dr. Stein et al ... 838
Schick, B.: Effect of tonsillectomy and adenoidectomy on diphtheria immunity. *929
Schick Reaction and circulating antitoxin .. 854
Schlatter-Osgood Disease: See Tibia, tuberosity
Schloss, O. M.: Pathology of so-called "acute pyelitis" in infants *227
School children, scholastic efficiency of, without full complement of parents 1109
hygiene, 6 months of 1328
medical organization in Uruguay 1329
medical service in Buenos Aires, evolution of 1329
medical service in Chile 1328
Schumm, H. C.: Juvenile flat feet *1273
Schwartz, S. P.: Digitalis studies on children with heart disease; effects of digitalis on electrocardiograms of children with rheumatic fever and chronic valvular heart disease *699
Schwarz, H.: Bacteremia and skin manifestations in lipoid nephrosis *762
"Blood cultures after tonsillectomy" *1282
Sclerema neonatorum 1297
Scleroderma and calcinosis in a child treated by ketogenic diet 603
extensive, progeria beginning with 643
familial 1323
in new-born 450
Scoliosis: See under Spine
Scurvy, experimental, minute morphology, in guinea-pig 153
experimental, some characteristics of blood of guinea-pig in 154
experimental, symptomatology and gross morphology of 152
incipient 401

Scurvy—Continued PAGE
infantile, osteochondritis luetica, comparison with analogous zones in rickets and ... 622
studies on ... 152
Sea climate, psychologic investigations on effect of ... 425
Septicemia and pericarditis due to streptococcus ... 859
Serum and vaccine in prevention of disease 664
and vaccine therapy in pediatrics ... 1325
anti-vaccinal ... 843
calf, use of, in pediatrics ... 647
determination of value of, in treatment for meningococcus meningitis ... *730
disease, treatment of ... 419
horse, and mecurachrone, study of action of, on pneumococcus infections in mice. 596
horse, ophthalmic test for sensitiveness to ... 171
Shaw, H. L. K.: The American pediatric society and preventive pediatrics ... *1
Sheplar, A.: Determination of value of serum in treatment of meningococcus meningitis; report of illustrative series of cases ... *730
Sherman, I.: Congenital essential thrombocytopenia ... *1245
Shoes for the child ... 645
Shohl, A. T.: Preparation of dextrose for intraperitoneal injection ... *943
Preparation of sodium bicarbonate for intraperitoneal use ... *927
Shyness ... 177
Silver, H. B.: Acute mastoiditis in infant aged 8 weeks ... *567
Silver Nitrate solution, serious hemorrhage from conjunctiva in infant following use of ... 186
Sinus, maxillary: See Antrum
sinusitis in infancy and childhood ... 1106
Skin, action of ultraviolet rays on ... 200
cutaneous induration in new-born infant 162
diseases caused by internal disturbances. 882
diseases, rôle of allergy and idiosyncrasy in ... 1322
disseminated tuberculosis, verrucosa cutis following measles ... 1323
psychic and nervous component of so-called allergic skin diseases ... 199
tuberculosis (Besredka's method) complement fixation in ... 149
Sleep, narcolepsies ... 423
Smallpox ... 1082
generalized vaccinia in rabbits with internal lesions ... 148
localization of cowpox vaccine in blood of vaccinated guinea pig ... 599
protection against, in France ... 409
vaccination and Dick reaction ... 620
variola-vaccinia ... 618
Smith, C. H.: Tuberculin skin reactions .. *1137
Sneezing, paroxysmal, as equivalent of paroxysmal cough of pertussis ... 612
Societies: Canadian societies, directory of ... 223, 447, 679, 901
committee for United States for Second International Pediatric Congress ... 1280
Couzens Children's Fund of Michigan holds conference ... 1281
international, directory of ... 447, 679, 901, 1121, 1351
local, directory of ... 223, 447, 679, 901, 1121, 1351
national, directory of ... 223, 447, 679, 901, 1121, 1351
Second International Pediatric Congress in Stockholm, August 17-20, 1930 ... 1280
Spanish Association for Progress of Science, 12th congress of ... 1329
University of Michigan Pediatric and Infectious Disease Society ... 1280
Society Transactions:
American Pediatric Society ... 432, 648
Central States Pediatric Society ... 1330
Chicago Pediatric Society .. 203, 207, 671, 891
Cleveland Academy of Medicine, Section on Pediatrics ... 676
New York Academy of Medicine, Section of Pediatrics ... 210, 669, 1111
Philadelphia Pediatric Society ... 219
Sodium bicarbonate, preparation of, for intraperitoneal use ... *927
chloride, alterations in acid base metabolism in alimentary and in sodium chloride fever ... 403
Sorbite as source of carbohydrate in diabetes ... 420
Spasm, ankle-clonus; distinction of organic and functional varieties ... 424
oculogyric crises in chronic epidemic encephalitis ... 424
Spasmophilia, case report ... 896
manifest, ergosterin therapy in ... 637
rachitic, and human rickets, quartz lamp therapy in ... 602
Speech, development of ... 422
inner ... 637
Spinal Cord, tumor like nodule in lateral wall of, in human embryo of 16.5 cubic millimeters ... 159
Spine, Klippel-Feil syndrome with marked vertebral anomalies; report of case ... 886
muscular atrophy of Werdrig-Hoffmann and amyotonia congenita in 1 family .. 185
Pott's disease with paraplegia ... 887
scoliosis and its treatment ... 1108
tuberculosis, rupture of cold abscesses in, into pleuro-pulmonary passages and into digestive tract ... 1304
tumor, epigastralgic syndrome of vertebral and intravertebral origin ... 633
Spleen, function of, in organic immunity ... 1075
histologic changes in, in early congenital syphilis ... 1088
puncture, as diagnostic procedure in infancy and childhood ... 655
Splenectomy for sickle-cell anemia ... 1323
thrombocytopenia purpura treated by repeated blood transfusions and ... 672
Splenohepatomegaly: See under Niemann's Disease
Splenomegaly: See also Niemann's Disease
a symposium ... 863
hepatic cirrhosis with ascites and, under anti-syphilitic treatment ... 624
portal phleboscierosis ... *1231
Sputum, smears from larynx ... 414
Stammering, constitutional studies of stammerers ... 1107
Status Thymicolymphaticus: See Lymphatism
Stein, I. F.: Reply to comment of Dr. Schauffler ... 838
Steiner, M.: Diaphragmatic hernia in infancy and in childhood ... *361
Step Parent, the ... 636
Sternum, absence of xiphoid cartilage and its value in diagnosis of congenital syphilis ... 169
Stoesser, A. V.: Solitary nonparasitic cysts of liver ... *241
Stoloff, E. G.: Pathologic changes in large interlobar fissures in children ... *75
Stomach, cardiospasm and pylorospasm ... 1307
cardiospasm in infancy and in childhood. *294
cardiospasm in new-born infant, report of 3 cases ... *1183
radium capsule in; gastroscopic removal .. 1093
rupture, acute ... 1339
ulcers, perforating ... 1101
Stomatitis, aphthous ... 1340
chronic mycelial, and chronic tetany in child aged 4½ years ... *328
Streptococcus cardioarthritis, biologic and serologic studies of ... 147
hemolytic, and scarlet fever ... 411
in throats of children ... 882
infection of tonsils in varicella complicated by acute nephritis ... 852
mucosus infection ... 1098
organisms associated with commercially prepared infant foods ... *310
pericarditis and septicemias due to ... 859
relation of, to spinal fluid in experimental poliomyelitis ... 596
Stridor, congenital, cysts of tongue in ... 428
congenital, of new-born, phenocoll hydrochloride in ... 1082

PAGE

Stuttering, study of simultaneous antitropic movements of hands of stutterers.....1312
Subarachnoid space, anatomy of, cerebrospinal fluid and barrier between blood and spinal fluid..... 1284
Sugar, problem of sweets for children..... 400
Sullivan, R. C.: Carbohydrate tolerance in infants and in young children with celiac disease..... *16
Sulphur in blood of children..... 600
in human and cow's milk..... 605
Suprarenal capsule in tuberculosis.....1089
cephalic metastases of suprarenal blastomata in children..... 172
hemorrhage 1331
reported hypoplasia of, in so-called status thymicolymphaticus 1097
Sweat and Sweat Glands, basal insensible perspiration of infants..... 436
Syncope, paroxysmal tachycardia with, in a child *551
Synostosis: See Abnormalities
Synthalin in juvenile diabetes.....863
Syphilis: See also Neurosyphilis
and pregnancy 1301
bone disease in.....1092
congenital 1345
congenital, absence of xiphoid cartilage and its value in diagnosis of..... 169
congenital, and juvenile psychopathies...1089
congenital, arsenical reactions in, complicated with alkalosis..... 623
congenital, destructive changes in bony shafts in.....1305
congenital, diagnosis by Lange reaction..1304
congenital, histologic changes in spleen in 1088
congenital, in children; intraperitoneal injections of neosalvarsan and mercuroxal in..... 412
congenital, incidence of, among dependent children 218
congenital, malaria treatment for...414, 1305
congenital, muscular alterations observed in osteopathies of infancy..... 413
congenital, teeth in.....1336
congenital, treatment for.....1305
congenital, unilateral optic atrophy in..1304
congenital, unusual skin eruption in..170, 623
congenital Wassermann-fast, in 2 sisters 170
hereditary, infantile hemiplegia and mental retardation in.....1089
hereditary, of liver, anatomic forms of..1088
Kahn precipitation test for.....1115
Kahn precipitation test in; comparative study with Noguchi-Wassermann test..*1206
latent, flareups of, in children..... 170
management of syphilitic pregnant women; its relation to prognosis for the child 625
syphilitic hypotrophy.....1088

Tachycardia: See Heart, rate
Tapeworm in children; treatment..... 633
Teeth, abnormal dentition.....1326
caries of, and diet.....439, *721
"circular caries" of deciduous teeth, pathogenesis of.....1309
effect of antenatal and postnatal diet on structure of, and incidence of dental caries 154
effect of rickets and severe nutritional disturbances on..... 440
Hutchinsonian, association and pathogenesis 168
in congenital syphilis.....1336
malformations and caries.....1337
unerupted 646
Temperature records, continuous, of premature infants *944
Tendon, Achilles, clinical value of sensitivity to pressure of, and of testicle... 426
Teratoma, mediastinal, in young infant.... 858
Testicle, clinical value of sensitivity to pressure of..... 426
undescended, and inguinal hernia in single ovum twins.....1078
Tetanus, case of, simulating tetany..... 408
postvaccinal, rôle of vaccination dressing in production of..... 853
Tetany and rickets in rats..... 145

Tetany—Continued PAGE
and water economy; relations between... 637
case of tetanus simulating..... 408
cataract and dwarfism..... 190
chronic, and chronic mycelial stomatitis in child aged 4½ years.....*328
Chvostek sign in infants, effect of outdoor schools and sunbathing on..... 180
effect of ligation of common bile duct upon appearance of, in thyroparathyroidectomized dogs..... 144
irradiated ergosterol in..... 422
sex, age and seasonal distribution of, in orphanages in Peking..... 421
Thallium acetate epilation in tinea capitis..1323
acetate, poisoning by..... 883
medication, in tinea capitis and other dermatoses requiring epilation..... 642
Thirst, question of polydipsia..... 868
Thoracectomy, precordial, for adherent pericardium 886
Thoracopagi 1296
Thoracoplasty in pulmonary tuberculosis..1093
Thorax, interpretation of roentgenograms of chest based on necropsies.....
.....208, *978, *1166
simulation of pneumothorax by thoracic rickets 402
successive roentgenograms of chest during measles *258, 1118
Thorpe, E. S., Jr.: Chronic tetany and chronic mycelial stomatitis in child aged 4½ years.....*328
Throat, infection, leukocytic response to... 881
streptococci infection in..... 882
Thrombocytopenia, essential, congenital...*1245
Thrombopenia, rate of disintegration of blood platelets in normal children in hemophilia and..... 600
Thumb, painless swelling of..... 889
Thymus and rickets..... 672
histologic changes, in children with scarlet fever..... 617
involution and regeneration under influence of benzene..... 844
preparation for fragilitas ossium.....1100
significance of concentric corpuscles of Hassall 143
some sidelights on the thymus vogue..... 631
Thyroid, blood supply of, with reference to vascular system of cretin goiter..... 843
changes in, in avitaminosis B..... 845
extract, influence of, on protein and fat metabolism 599
extract, standardization of..... 430
of cretins, pathologic anatomy of malignant goiter and..... 867
preparations in myxedema.....1312
Thyroxin in infantile myxedema..... 421
Tibia, bilateral osteomyelitis of..... 885
Osgood-Schlatter's disease.....884, 1108
Tinea capitis, thallium medication in..642, 1323
Tissue changes associated with vitamin "A" deficiency in the rat..... 844
Tokue, K.: Agranulocytosis; first case reported in Japan.....*1037
Tongue, cysts of, in congenital stridor.... 428
Tonsillectomy and rheumatic fever..... 853
and scarlet fever..... 164
blood cultures after.....*726, *1282
effect of adenoidectomy and, on diphtheria immunity 432, *929
efficacy of, for removal of focal infections 639
indications for.....1106
Tonsillitis: See Tonsils
Tonsils and nutrition.....1344
diseased, use of radium in..... 639
tuberculosis, primary.....1305
vessel changes in peritonsillar tissue in chronic tonsillitis..... 196
Toomey, J. A.: Studies in scarlet fever; blanching with placental serum.....*953
Topper, A.: Basal metabolism in children of abnormal body weight in underweight children *299
Effect of tonsillectomy and adenoidectomy on diphtheria immunity.....*929
Torticollis (caput obstipum).....1078
congenital 159
removal in early life of fibrous mass from sternomastoid muscle..... 885
study of.....1108

PAGE

Toxicosis, blood serum phosphate in..... 599
increase of, among infants in Germany during the summer of 1928..... 605
Toxin and Antitoxin, report of the royal commission of inquiry into fatalities from toxin-antitoxin at Bundaberg.... 614
Toxoid for immunization against diphtheria *35, 617, 1084, 1332
for immunization against scarlet fever....1332
Trachea, congenital atresia of esophagus with tracheal anastomosis.....1296
congenital atresia of esophagus with tracheo-esophageal fistula..... 404
foreign body in, simulating diphtheria... *95
papillomatosis of larynx and..... 639
Tracheobronchial glands: See under Lymph Nodes
Tracheotomy for acute laryngitis with edema 882
Trauma, birth, as cause of diffuse dilatation of esophagus..... 159
birth, of central nervous system..... 405
Trichocephalus dispar, acetarsone treatment of.....1099
Trichophyton infections, allergy and immunity in.....1322
Tuberculin skin reactions.....*1137
studies 1093
tests, size of reacting area in intracutaneous tests in relation to classification of disease and other clinical factors...*1155
Tuberculoma, solitary, unusual location of. 870
Tuberculosis, allergy, immunity and other factors of importance in pathogenesis of 1302
and erythema nodosum.....1107
bacillus, transplacental infection with tuberculous ultravirus and tuberculous heredity 169
bacteriologic and experimental study of pus from abscesses following injections of B. C. G..... 414
blood picture of, in childhood..... 170
Calmette-Guérin bacillus experimental researches on necrotic lesions provoked by injection of massive doses of..... 169
Calmette-Guérin bacillus, experimental study of B. C. G.....1090
Calmette-Guérin bacilli, reinfection of tuberculous guinea-pigs and guinea-pigs immunized with..... 169
Calmette-Guérin bacillus vaccination of infants 413
Calmette-Guérin bacillus, virulence of...1093
corticopleural 1090
Costa reaction in, in children..... 626
cutis: See under Skin
foreign bodies causing changes simulating 1091
in childhood.....167, 1344
in childhood, fundamental importance of extrafamilial infection with, in childhood 625
in children from sociologic point of view.1346
in infants under 1 year of age; study of autopsy and clinical observations on infants with tuberculous lesions.....*526
incidence of tuberculous infection in school children..... 622
Langer's method of vaccination..... 625
lesions in lung of negro child 9 weeks old 412
mortality in infants with familial exposure to 413
of abdominal lymph nodes.....1088
of tracheobronchial gland with anatomic control 648
of tracheobronchial glands, diagnosis of.*1125
primary, of middle ear, in infants..... 193
psychic changes in children with..... 626
pulmonary, acute disseminated miliary, pathology of.....1091
pulmonary, in children.....1302
pulmonary, and pneumococcemia.....1090
pulmonary, incipient, principal types of; diagnosis through roentgenography and investigation of bacillus..... 624
pulmonary, interpretation of roentgenograms of chest based on observations at necropsy.....208, *978, *1166
pulmonary, roentgenography in incipient forms with anatomic controls.....1304

Tuberculosis—Continued PAGE
pulmonary, thoracoplasty in.....1093
report of study of 25,048 school children for 412
suprarenal capsule in.....1089
testicular allergic response to guinea-pig for diagnosis of tuberculous fluids..... 841
tonsillar, primary.....1305
tuberculous bronchopneumonia in infant. 170
Tularemia 1305
Tumor, cerebral, of infancy..... 180
of posterior fossa, encephalography in... 184
of spine, epigastralgic syndrome of vertebral and intravertebral origin..... 633
sacrococcygeal 174
Twins, microcephaly in.....1296
thoracopagi 1296
undescended testicle and inguinal hernia in single ovum twins.....1078
Tympanum: See Ear
Typhoid, brilliant-green enrichment methods in examination of feces for organisms of enteric group..... 147
enteric fever, 1895-1914.....1079
in children, study of 67 cases..... *10
in infant less than one month old.....*1044
Widal's reaction and complement fixation test in.....1085
Tyson, R. M.: Continuous temperature records of premature infants.....*944

Ultraviolet Rays, accidents in..... 647
action of, on skin..... 200
as aid in diagnosis of ringworm of scalp.*339
biologic effects of fats and chocolate irradiated by..... 606
correlation of ultraviolet absorption to development of immunity..... 668
effect of carbon arc radiation on blood of dogs 144
effect of irradiation on the antibody formation 601
effect of, on body and growth of child.155, 431
effect on new-born infant of irradiating the mother during pregnancy..... 611
influence of yeast, irradiated yeast and, on sugar tolerance of diabetic children...*513
penetration into live animal tissues..... 144
polymorphonuclear count in the newborn; deflection by ultraviolet rays...*547
quartz lamp therapy in human rickets and rachitic spasmophilia..... 602
relation of altitude of sun to its antirachitic effect 602
therapy for prevention of rickets..... 601
uses and abuses..... 888
Uranin: See Fluoresceine-Sodium
Uremia, true cause of.....1074
Ureter, obstruction in infancy..... 628
ureteral neuromuscular dystrophy in relation to infections of kidneys.....*1006
Urethra, congenital valves of posterior.....*804
male, diphtheria bacilli in..... 410
prostatic, congenital valvular obstruction of 846
prostatic, imperforate anus with exit through; report of 3 cases.....*559
Urinary Tract, anomalies of, frequency and types in a consecutive necropsy series.*960
anomalies in infants.....*1202
congenital dilatation of..... 608
disease, relation of spontaneous bacilluria and pyelitis to mode of infection in... .....432, *968
infection, in infants treated with colon bacillus antiserum of Vincent..... 858
upper, roentgen diagnosis of lesions unassociated with calculus..... 627
urologic conditions in children..... 675
Urine, blood in, refinement of benzidine test for 400
disturbances in infant caused by hexamethylenamine 419
pus, clinical discussion and therapy of pyuria 862
pyuria, chronic.....1307
pyuria, cystogram in.....1101
pyuria, persistent, in infants and children 215
spontaneous pyelitis and bacilluria in rabbit; relation to mode of infection in urinary tract disease.....432, *968
urobilinuria in heart disease.....1094

PAGE

Urobilinuria: See under Urine
Urticaria and allied disorders, cesium eosinate in 200
pigmentosa 200, 1331
pigmentosa; report of nodular case and review of literature.....*1020
Uterus, cancer of, microcephalic idiocy following radium therapy of, during pregnancy 1102

Vaccination, encephalitis following.184, 411, 1102
Vaccine and serum in prevention and treatment 664
and serum therapy in pediatrics.....1325
localization of cowpox vaccine in blood of vaccinated guinea pig..... 599
therapy and blood transfusion in diseases of children.....1327
virus in blood after cutaneous inoculation, demonstration of..... 150
Vaccinia: See under Smallpox
Vagina, biology of, in infants..... 400
method of treatment of diseases of, in young girls with unruptured hymen.... 861
Van Creveld, S., Carbohydrate metabolism of premature infants; blood sugar during fasting.....*912
Varicella: See Chickenpox
Variola: See Smallpox
Veins, femoral, congenital lipoma of..... 159
Venereal diseases, occurrence in children..1083
Viscera, transposition of, with congenital true dextrocardia.....1078
Vision, hemangioma of meninges involving visual cortex.....1313
inverted position in children's drawings; report of 2 cases..... 177
temporary loss of eyesight due to pressure of fluid 185
Vital Statistics, analysis of mortality figures of Willard Parker Hospital for years 1922-1927, inclusive..... 613
decrease in maternal and infant mortality in Oregon..... 888
infant mortality and birth rate in Bavaria 890
stillbirths and neonatal deaths in 1,960 consecutive private cases..... 450
summary of provisional death and infant mortality figures in birth registration area: 1928..... 888
Vitamin A deficiency in rat, tissue changes associated with..... 844
A in retinal tissue and comparison with vitamin content of brain tissue.....1074
avitaminosis B, changes in thyroid gland in 845
B 603
B and B_2, nervous system in rats fed on diets deficient in..... 845
B factor in infant feeding, routine use of 602
C, deficiency, association of bowel disease with 155
C, therapeutic use of.....1290
D, antirachitic, method of assay of.....1287
D, growth promoting properties of.....1288
dual nature of water-soluble vitamin B; effects upon young rats of vitamin B2 deficiency and method for biological assay of vitamin B 2.....1287
effect of heat and exposure to air on stability of; rickets in chicks.....*481
Volhard's stasis following concretio cordis.1310
Volvulus: See Intestines
Vomiting, cyclic, influence of emotional shock and rôle of nervous system in acidosis of.....1327
emesis in nursling, clinical considerations.1096
spontaneous hypoglycemia in childhood...*746
Vulvitis aphthosa..... 419
Vulvovaginitis, gonorrheal, malarial treatment in..... 616

PAGE

Wallgren, A.: Pathogenesis of acute serofibrinous pleurisy *829
Wang, C. C.: Metabolism of undernourished children; study of basal metabolism caloric balance and protein metabolism during a period of gain in weight.....*476
Metabolism of undernourished children; effect of high and low protein diets on excretion of creatine, creatinine and ammonia *468
Wangensteen, O. H.: Solitary nonparasitic cysts of liver.....*241
Waring, J. I.: Beriberi in infants..... *52
Wassermann Reaction, standardization of.. 412
Kahn precipitation test; a clinical comparison with Noguchi-Wassermann test *1206
Wedd, A. M.: Ectopia cordis.....*562
Weech, A. A.: Typhoid fever; report of case in infant less than 1 month of age....*1044
Weight: See under Body
Weinfeld, G. F.: Comparative effects of diphtheria toxoid and toxin-antitoxin as immunizing agents..... *35
Weiss, M. M.: Digitalis studies on children with heart disease; effects of digitalis on electrocardiograms of children with rheumatic fever and chronic valvular heart disease.....*699
Werner, M.: Blood cultures after tonsillectomy *726
White, P. J.: Relation between colic and eczema in early infancy.....*935
Whooping Cough..... 854
bacteriologic diagnosis of.....1299
blood picture in.....1084
early diagnosis of.....1301
encephalitis accompanying; clinical history and report of postmortem examination *97
encephalitis in.....615, 1340
epidemiology of..... 405
generalized emphysema in bronchopneumonia and..... 616
measles and chicken pox, completeness of reporting of, at different ages..... 614
paroxysmal sneezing as an equivalent of paroxysmal cough of pertussis..... 612
phenocoll hydrochloride in.....1082
prevention and treatment..... 407
prophylaxis and therapy in..... 617
vaccine in prophylaxis.....1343
vaccine therapy or early diagnosis..... 162
Wile, I. S.: Constipation and behavior.....*570
Willius, F. A.: Paroxysmal tachycardia with syncope occurring in a child.....*551
Wilson, J. R.: Pathology of so-called "acute pyelitis" in infants.....*227
Wilson, S. J.: Splenomegaly: Portal phleboscleresis *1231
Wood light in diagnosis of ringworm of scalp *339
Wright, I. S.: Determination of value of serum in treatment for meningococcus meningitis; report of illustrative series of cases.....*730
Wry-Neck: See Torticollis

Xiphoid Process: See Sternum

Yasumoto, M.: Agranulocytosis; first case reported in Japan.....*1037
Yater, W. M.: Congenital heart-block; review of literature; report of case with incomplete heterotaxy; the electrocardiogram in dextrocardia.....*112
Yeast, irradiated, and ultraviolet rays, influence of, on sugar tolerance of diabetic children.....*513

Zimmerman, H. M.: Encephalitis accompanying whooping cough; clinical history and report of postmortem examination *97

THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published by the American Medical Association as a means for the advancement of knowledge of the diseases of children and for the publication of scientific investigation in this field.

Manuscripts for publication, books for review, and correspondence relating to the editorial management should be sent to Dr. Clifford G. Grulee, 310 South Michigan Avenue, Chicago, or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, AMERICAN JOURNAL OF DISEASES OF CHILDREN, American Medical Association, 535 North Dearborn Street, Chicago.

Articles are accepted for publication on condition that they are contributed solely to the AMERICAN JOURNAL OF DISEASES OF CHILDREN. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones of illustrations will be supplied by the Association when the original illustrations warrant.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association. This requires, in order given: name of author, title of article, name of periodical, with volume, page, month—day of month if weekly—and year. A complete list of abbreviations for standard periodicals, together with a full discussion of the style of the A. M. A. publications, appears in The Art and Practice of Medical Writing, a comprehensive book on the preparation of medical manuscripts, published by the American Medical Association. Price, $1.50.

Matter appearing in the AMERICAN JOURNAL OF DISEASES OF CHILDREN is covered by copyright; but, as a rule, no objection will be made to its reproduction in reputable medical journals, if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the AMERICAN JOURNAL OF DISEASES OF CHILDREN or in any one of the other publications issued by the American Medical Association will not be permitted.

Authors will receive one hundred reprints free; additional reprints may be obtained at cost.

THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published monthly. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75, including postage. Single copies, 85 cents, postpaid.

Checks, etc., should be made payable to the AMERICAN MEDICAL ASSOCIATION.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (two volumes): Domestic, $7.00; Canadian, $8.50; foreign, $9.50. Single copies, 25 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced, original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): Domestic, $5.00; Canadian, $5.40; foreign, $5.75. Single copies, 75 cents.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, society transactions, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions and full abstracts of the literature on these two subjects, transactions of the important dermatological societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews on orthopedic and urologic surgery. Well illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF OTOLARYNGOLOGY—Monthly. A medium for the presentation of original articles on diseases of the ear, nose and throat, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $6.75. Single copies, 75 cents.

ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original contributions in the field of pathology, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $6.75. Single copies, 75 cents.

ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth-while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: Domestic, $12.00; Canadian, $12.50; foreign, $12.75

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn St. **CHICAGO**

CONTENTS

PAGE

THE DIAGNOSIS OF TUBERCULOSIS OF THE TRACHEOBRONCHIAL GLANDS. P. ARMAND-DELILLE, M.D., AND C. LESTOCQUOY, M.D., PARIS, FRANCE 1125

TUBERCULIN SKIN REACTIONS. CHARLES HENDEE SMITH, M.D., NEW YORK 1137

THE SIZE OF THE REACTING AREA IN INTRACUTANEOUS TUBERCULIN TESTS IN RELATION TO CLASSIFICATION OF DISEASE AND TO OTHER CLINICAL FACTORS. LLOYD B. DICKEY, M.D., SAN FRANCISCO 1155

INTERPRETATION OF ROENTGENOGRAMS OF THE CHEST IN CHILDREN BASED ON OBSERVATIONS AT NECROPSY: II. TUBERCULOSIS. JOHN A. BIGLER, M.D., CHICAGO 1166

CARDIOSPASM IN THE NEW-BORN INFANT: REPORT OF THREE CASES. T. L. BIRNBERG, M.D., ST. PAUL 1183

INTRACRANIAL HEMORRHAGE IN THE NEW-BORN INFANT AS DEMONSTRATED BY THE ROENTGEN RAYS. M. HINES ROBERTS, M.D., ATLANTA, GA. 1196

ANOMALIES OF THE URINARY TRACT IN INFANTS. ALGERNON S. HURT, JR., M.D., ROCHESTER, MINN. 1202

THE KAHN PRECIPITATION TEST IN INFANCY AND IN EARLY CHILDHOOD: A COMPARATIVE CLINICAL STUDY WITH THE NOGUCHI-WASSERMANN REACTION. JOHN CAFFEY, M.D., AND KATHERINE V. KREIDEL, A.B., NEW YORK 1206

THE RELATIONS OF WEIGHT, HEIGHT AND AGE IN NEGRO CHILDREN. L. T. ROYSTER, M.D., AND C. N. HULVEY, M.S., L.L.B., UNIVERSITY OF VIRGINIA, VA. 1222

CASE REPORTS:

SPLENOMEGALY: PORTAL PHLEBOSCLEROSIS. SAMPSON J. WILSON, M.D., AND MAX LEDERER, M.D., BROOKLYN 1231

CONGENITAL ESSENTIAL THROMBOCYTOPENIA. HARRY M. GREENWALD, M.D., AND IRVING SHERMAN, M.D., BROOKLYN 1245

PNEUMONIC ANTHRAX IN A CHILD. J. C. MCKITTRICK, M.D., AND GEORGE J. PEARSON, M.D., BURLINGTON, IOWA 1252

PROGRESS IN PEDIATRICS:

BILATERAL CONGENITAL FACIAL PARALYSIS: REVIEW OF THE LITERATURE AND A CLASSIFICATION. BARNET E. BONAR, M.D., SALT LAKE CITY, AND RUSSELL W. OWENS, M.D., MAGNA, UTAH 1256

JUVENILE FLAT FEET. H. C. SCHUMM, M.D., MILWAUKEE 1273

PEDIATRIC BIOGRAPHIES:

J. STARSMARE. JOHN RUHRÄH, M.D., BALTIMORE 1277

NEWS AND COMMENT 1280

CORRESPONDENCE:

BLOOD CULTURES AFTER TONSILLECTOMY. HERMAN SCHWARZ, M.D., AND IRVING A. FRISCH, M.D., NEW YORK 1282

ABSTRACTS FROM CURRENT LITERATURE 1283

SOCIETY TRANSACTIONS:

CENTRAL STATES PEDIATRIC SOCIETY 1330

BOOK REVIEWS 1348

DIRECTORY OF PEDIATRIC SOCIETIES 1351

GENERAL INDEX 1355